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INDOOR AIR POLLUTION

IN MASSACHUSETTS

Interim Report

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The Commonwealth of Massachusetts
Special Legislative Commission
on Indoor Air Pollution

June 1988

883/271

INDOOR AIR POLLUTION

IN MASSACHUSETTS

INTERIM REPORT OF

THE SPECIAL LEGISLATIVE COMMISSION

ON INDOOR AIR POLLUTION

**Under Chapter 10 of the Resolves of 1986
and Chapter 2 of the Resolves of 1987**

Prepared by:

Sally A. Zielinski, Ph.D.

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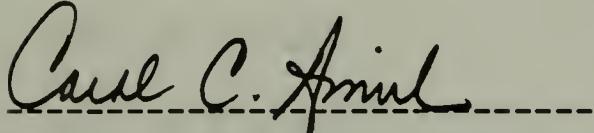
**Office of Senator Carol C. Amick
June 1988**

LETTER OF TRANSMITTAL

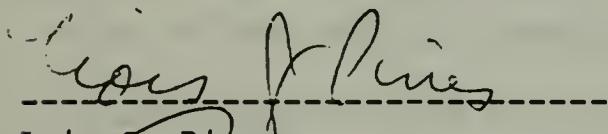
To the Honorable Senate and the House of Representatives:

We, the undersigned, having voted in the affirmative, do hereby transmit this interim report on the results of the investigation and study authorized under the provisions of Chapter 10 of the Resolves of 1986 and Chapter 2 of the Resolves of 1987.

Respectfully Submitted,



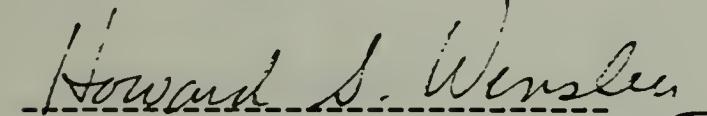
Carol C. Amick, Senate Chair



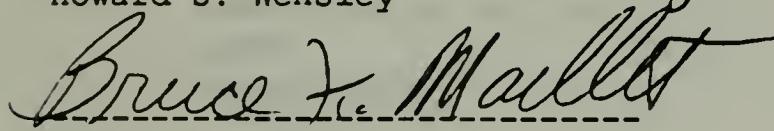
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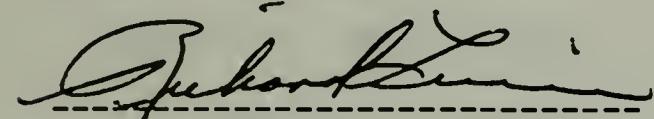
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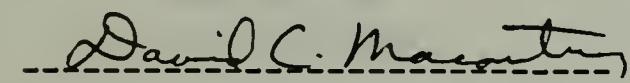
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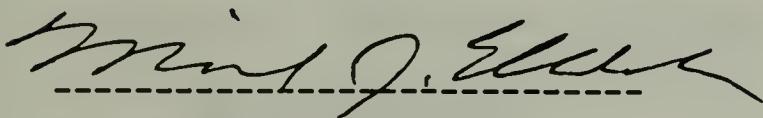
Bruce K. Maillet



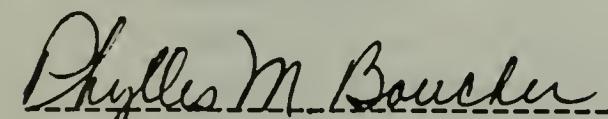
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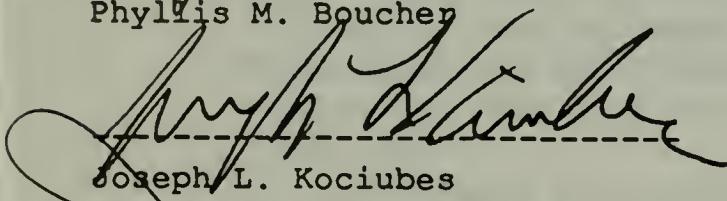
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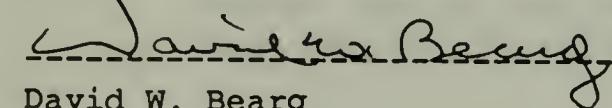
Michael Ellenbecker



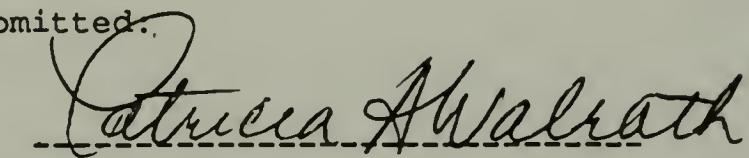
Phyllis M. Boucher



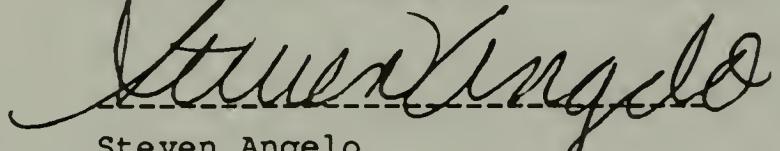
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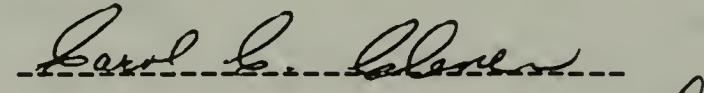
David W. Bearg



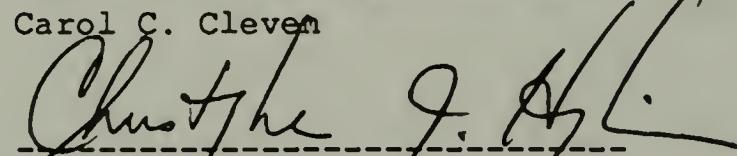
Patricia A. Walrath, House Chair



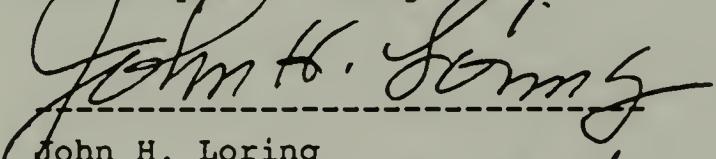
Steven Angelo



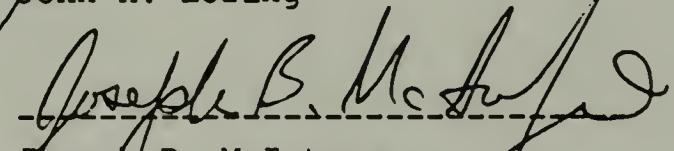
Carol C. Cleven



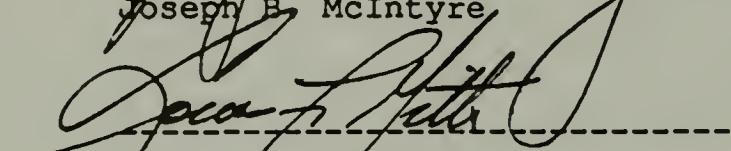
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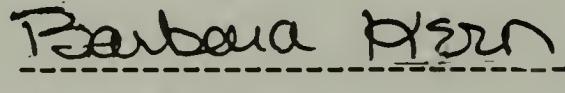
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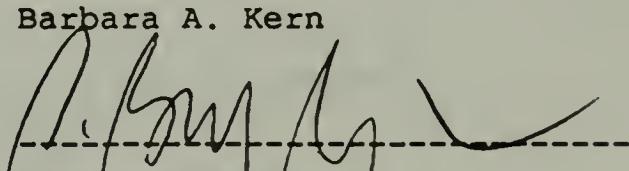
Joseph B. McIntyre



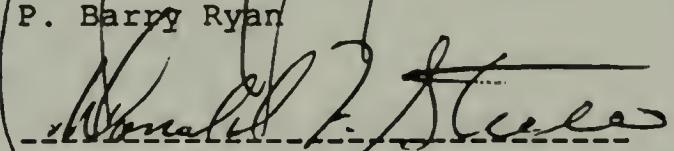
Louis F. Gitto



Barbara A. Kern



P. Barry Ryan



Donald F. Steele

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LIST OF ACRONYMS AND TECHNICAL TERMS

ach	air changes per hour
AHERA	Asbestos Hazard Emergency Response Act
AIHA	American Industrial Hygiene Association
ASHRAE	American Society of Heating, Refrigeration and Air Conditioning Engineers
BOH	Massachusetts local Board of Health
BPA	Bonneville Power Administration
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act (Superfund)
cfm	cubic feet per minute
CIAQ	Committee on Indoor Air Quality
CIIT	Chemical Industry Institute of Toxicology
CPSC	Consumer Product Safety Commission
DEQE	Massachusetts Department of Environmental Quality Engineering
DHHS	Department of Health and Human Services
DLI	Massachusetts Department of Labor and Industries
DNA	deoxyribonucleic acid
DOE	Department of Energy
DPH	Massachusetts Department of Public Health
EPA	Environmental Protection Agency
FDA	Food and Drug Administration
FEMA	Federal Emergency Management Agency
FHA	Federal Housing Administration
FIFRA	Federal Insecticide Fungicide and Rodenticide Act
FTC	Federal Trade Commission
HUD	Department of Housing and Urban Development
HVAC	Heating, ventilating and air conditioning
IRIS	Integrated Risk Information System
MDF	medium density fiberboard
NAAQS	National ambient air quality standards
NAS	National Academy of Sciences
NCI	National Cancer Institute
NCRP	National Council on Radiation Protection and Measurement
NIH	National Institutes of Health
NIOSH	National Institute of Occupational Safety and Health
NRC	National Research Council
OHER	Office of Health and Environmental Research
OSHA	Occupational Safety and Health Administration
pCi/l	pico Curies per liter
PF	phenol formaldehyde
ppm	parts per million
RCP	Massachusetts Department of Public Health, Radiation Control Project
RMP	Radon/Radon Progeny Measurement Proficiency
SARA	Superfund Amendment and Reauthorization Act
SBS	sick building syndrome
SDWA	Safe Drinking Water Act

TSCA	Toxic Substances Control Act
UF	urea formaldehyde
UFFI	urea formaldehyde foam insulation
UMTRCA	Uranium Mill Tailings Radiation Control Act
VOC	volatile organic compound
WL	working level
WLM	working level month

RESOLVE CREATING THE COMMISSION

H 6266

Chapter 10

THE COMMONWEALTH OF MASSACHUSETTS

In the Year One Thousand Nine Hundred and Eighty-six

RESOLVE PROVIDING FOR AN INVESTIGATION AND STUDY OF THE PUBLIC HEALTH EFFECTS OF INDOOR AIR POLLUTION.

RESOLVED, That a special commission consisting of four members of the house of representatives, three members of the senate, the commissioner of the department of public health or his designee, the commissioner of the department of environmental quality engineering or his designee, the commissioner of the department of labor and industries or his designee, the chairman of the state board of building regulations or his designee, the Regional Administrator (Region I) of the U.S. Environmental Protection Agency or his designee and seven persons to be appointed by the governor, one of whom shall be a member of the American Lung Association of Massachusetts, one of whom shall be a member of the Massachusetts Association of Health Boards, one of whom shall be a member of the Massachusetts Health Officers Association, one of whom shall be an academician from a local college or university with expertise in the area of indoor air pollution and mitigating its effects, one of whom shall be representative of the building materials industry, one of whom shall be a representative of the heating and ventilating industry, and one of whom has expertise in the area of indoor air pollution mitigation, is hereby established for the purpose of making an investigation and study of the public health effects of indoor air pollution, so called, including but not limited to, the effects of natural emissions of radon, the emissions from building materials such as urea formaldehyde and asbestos, the effects of emissions from wood stove, coal stoves, and fireplaces and the relationship between ambient air pollution and indoor air pollution. The commission shall also study the effects of personal habits, such as smoking and the relationship of indoor air pollution and ener-

gy conservation measures. Said commission shall report to the general court the results of its investigation and study, and its recommendations, if any, together with drafts of legislation necessary to carry such recommendations into effect by filing the same with the clerk of the house of representatives on or before the first Wednesday in November, nineteen hundred and eighty-seven.

House of Representatives, December 11, 1986.

Passed,

George K. Lunn, Speaker.

In Senate, December 11, 1986.

Passed,

William F. Bulger, President.

December 23, 1986.

Approved,

Michael J. Dukakis, Governor.

RESOLVE INCREASING THE MEMBERSHIP OF THE COMMISSION

H 4812

Chapter 2.

THE COMMONWEALTH OF MASSACHUSETTS

In the Year One Thousand Nine Hundred and Eighty-seven

RESOLVE INCREASING THE MEMBERSHIP OF THE SPECIAL COMMISSION ESTABLISHED TO MAKE AN INVESTIGATION AND STUDY OF THE PUBLIC HEALTH EFFECTS OF INDOOR AIR POLLUTION.

RESOLVED, That the membership of the special commission, established by chapter ten of the resolves of nineteen hundred and eighty-six, is hereby increased by two members of the house of representatives and one member of the senate.

House of Representatives, July 9, 1987.

Passed,

George K. Lunn, Speaker.

In Senate, July 9, 1987.

Passed,

Walter J. Boenin, Acting President.

July 21, 1987.

Approved,

Michael J. Dukakis, Governor.

MEMBERSHIP OF THE COMMISSION

Appointed by the President:

Senator Carol C. Amick, Senate Chair
Senator Lois G. Pines
Senator Peter C. Webber

Appointed by the Speaker:

Representative Patricia A. Walrath, House Chair
Representative Steven Angelo
Representative Carol C. Cleven
Representative Christopher J. Hodgkins
Representative John H. Loring
Representative Joseph B. McIntyre

Howard S. Wensley	- Department of Public Health (designee of the Commissioner of Public Health)
Bruce K. Maillet	- Department of Environmental Quality Engineering (designee of the Commissioner of Environmental Quality Engineering)
Richard Levine	- Department of Labor and Industries (designee of the Commissioner of Labor and Industries)
David C. Macartney	- State Board of Building Regulations (designee of the Chairman of the State Board of Building Regulations)
Louis F. Gitto	- Environmental Protection Agency (designee of the Regional Administrator (Region 1) of the United States Environmental Protection Agency)

Appointed by the Governor:

Michael Ellenbecker	- American Lung Association (member of the American Lung Association of Massachusetts)
Barbara A. Kern	- Massachusetts Association of Health Boards (member of the Massachusetts Association of Health Boards)
Phyllis M. Boucher	- Massachusetts Health Officers Association (member of the Massachusetts Health Officers Association)
P. Barry Ryan	- Harvard School of Public Health (academician from a local college or university with expertise in the area of indoor air pollution and mitigating its effects)
Joseph L. Kociubes	- Bingham, Dana and Gould (representative of the building materials industry)
Donald F. Steele	- Airxchange, Inc. (representative of the heating and ventilating industry)
David W. Bearg	- Life Energy Associates (expertise in indoor air pollution mitigation)

* * *

Research Director: Elizabeth C. Conklin

EXECUTIVE SUMMARY

INDOOR AIR POLLUTION

Air is a mixture of gases, vapors and solid particles originating from natural processes or human activities. Air is regarded as polluted when one or more components of air occur at concentrations which negatively affect human health or the environment.

The air inside structures is known to contain numerous pollutants, e.g., tobacco smoke, pesticide residues, allergens, microbes, radon, asbestos, and formaldehyde. Depending upon concentrations and length of exposure, these have effects ranging from annoying to deadly. These pollutants are often found in greater concentrations indoors than outdoors.

Indoor air pollution is a growing problem in the United States, accounting for up to 50% of all illnesses and at an annual cost of up to 100 billion dollars. Energy conservation measures instituted in buildings following increased energy costs in the early 1970's add to these estimates. More insulation and tighter construction led to lower ventilation rates and buildup of contaminants. Many "sick" buildings have been identified where significant numbers of occupants suffer severe or recurring discomforts such as headache, eye irritation and respiratory problems. Specific conditions attributable to airborne contaminants include: cancer; heart, circulatory, and respiratory problems; birth defects; and mental, nervous, and immunological disorders.

Developing a risk profile for an indoor air pollutant includes calculating the probability of a given health effect arising in an exposed population. Risk profiles are based on: the hazardous nature of the substance; how the health effect varies with the concentration or total exposure to the substance; and whether concentrations or total exposures leading to the health effect actually occur in indoor air.

High levels of indoor air pollution can be mitigated in two general ways: through eliminating or controlling the source or by removing or diluting the contaminants in the air. For example, a tobacco smoke problem may be mitigated in a home by not permitting people to smoke or by increasing the air exchange rate to the point where contaminants exist at a tolerable level.

Policy decisions surrounding regulation of indoor air are complex but key to determining government's role in improving indoor air quality. At both the federal level and in Massachusetts authority over indoor air quality is fragmentary and spread across a variety of agencies.

RADON

Radon gas emitted from uranium-bearing rock and soil is associated with lung cancer in miners. Concern about health risks arose when elevated levels of radon were found inside homes built on mine wastes and later in homes on

undisturbed land. Levels of indoor radon are a function of several factors including the amount of uranium in the local substrate, other soil characteristics especially permeability, the pathways by which radon enters the structure and the living patterns of the residents. Radon can enter buildings: as a gas from the soil via gaps in the structure; from tap water which has passed through uranium-bearing rock; or in uranium-containing construction materials such as the stone in a fireplace.

Indoor radon at concentrations above the Environmental Protection Agency's (EPA) action level of 4 pico Curies per liter (pCi/l) annual average exposure has been found in many states, including Massachusetts. The bedrock geology of the Commonwealth indicates the potential for moderate to high radon levels in some areas. In others the rock type is usually low in uranium content but may have local hot spots. Several thousand measurements from homes in Massachusetts suggest that around 20% of homes could have the potential to exceed the action level. Though these were not all random samples the results, coupled with the Commonwealth's geological makeup, indicate the need for a firm indoor radon database. Through the efforts of this Commission funding was obtained for a radon study jointly conducted by EPA and the Department of Public Health (DPH). This study is now underway. It appears that elevated radon levels can be mitigated through modifications in structures, ventilation and living patterns.

The risk of dying of lung cancer from long term low level radon exposure is based both on the health effects observed at around 4 pCi/l and extrapolation from the effects of the high doses received by miners. A few structures have levels comparable to mines. Limited data suggests low doses are a hazard.

Lack of a comprehensive program at either the state or local levels makes action to address indoor radon a matter of some urgency.

FORMALDEHYDE

Formaldehyde is a reactive gas with a pungent odor which has been associated with health effects ranging from irritation of the eyes, skin and respiratory system, headache and nausea to potential cancer. Formaldehyde causes nasal cancer in animals and there is limited evidence of its carcinogenic effect in humans. EPA has designated formaldehyde a probable human carcinogen.

Formaldehyde is ubiquitous in the modern indoor environment. Its major source in indoor air is bonded wood products made with urea formaldehyde resins. Plywood and particleboard are in wide use in furniture, cabinets, flooring and wall paneling. Formaldehyde is also utilized in urea formaldehyde foam insulation (UFFI) and to treat textiles used for clothing, draperies and upholstery for crease resistance, colorfastness, shrink resistance, water and flame-proofing. Formaldehyde is a product of hydrocarbon combustion and therefore occurs in indoor air where stoves and heaters are improperly vented or people smoke tobacco products.

Elevated levels of formaldehyde may occur in mobile homes which contain high quantities of bonded wood products and are poorly ventilated, in UFFI homes, and in tightly constructed or new homes or in biological laboratories and

industrial facilities.

The Occupational Safety and Health Administration (OSHA) has reduced its workplace standard for an 8-hour average exposure from 3 to 1 ppm. The National Academy of Sciences (NAS) feels most healthy adults suffer no irritating effects at 0.25 ppm, however, the American Society of Heating, Refrigeration and Air Conditioning Engineers (ASHRAE) has adopted 0.1 ppm as a comfort based general indoor formaldehyde air quality guideline and the American Industrial Hygiene Association (AIHA) also recommends 0.1 ppm.

Formaldehyde concentration in a room is determined by the nature of the emitting material, its age, temperature, humidity, and ventilation. The Housing and Urban Development Agency (HUD) has developed product emission standards for bonded wood products in manufactured homes which are based upon assumptions regarding home design and ventilation. UFFI, which was sprayed into the walls of over 400,000 homes nationally during the 1970's, led to many complaints. While a federal Consumer Product Safety Commission (CPSC) ban was overturned in court UFFI is seldom used today. Massachusetts banned UFFI in 1979. Legislation passed in 1985 established disclosure, testing and removal programs funded by industry contributions. There are some administrative funds available from the state.

Chapter 1: INTRODUCTION

OVERVIEW OF PROBLEM

Indoor air quality is an emerging issue in Massachusetts, throughout the United States and in other industrialized countries. Reports of high radon levels in homes, non-smokers inhaling other people's tobacco smoke, sick buildings, and Legionnaire's disease mysteriously killing guests at a convention hotel, have drawn attention to the indoor environment.

Over the past several decades new substances have been identified in indoor air. Others have been found in unexpected concentrations. Higher levels of the same contaminant are often present indoors than outside, indicating that structures are no protection against air pollution. Most of these airborne compounds are colorless and odorless - undetectable to the senses.

Health effects associated with substances found in indoor air range from discomfort, such as nausea, headaches, eye and skin irritation, to chronic respiratory problems, cancer and mortality. For example, radon, formaldehyde and some components of tobacco smoke are suspected or proven carcinogens, and carbon monoxide from poorly vented stoves or heaters can cause brain dysfunction at low doses and death at high doses.

Since 90% of the average person's time is spent indoors, air quality in buildings is a public health issue which concerns not only owners and occupants but also government which must address a growing concern and develop approaches to improve indoor air quality.

GOALS

The overall goal of the Special Commission on Indoor Air Pollution is to develop a comprehensive approach to indoor air quality problems for the Commonwealth. Neither the federal government nor any state except California has a clearly structured, well coordinated program.

In response to the apparent magnitude and pervasiveness of indoor air pollution, it is the intent of this Commission to provide the legislature with the information and, if needed, legislation to protect the public from the adverse health effects of air pollution by:

- (1) establishing guidelines to achieve and maintain indoor air quality, and which address interrelationships among pollutants as well as individual pollutants
- (2) designating a comprehensive program for indoor air quality
- (3) increasing public awareness of the dangers from and solutions to indoor air pollution.

ACTION PLAN

The Commission's approach to this complex topic is to: investigate indoor air

quality as a whole; assess individual categories of pollutants then the interactive effects of multiple contaminants; then identify and address policy issues.

The many hazardous airborne gases, vapors and particulates found in indoor air have been grouped for convenience into the following categories: radon, formaldehyde and other volatile organic compounds (VOC's)*, asbestos, tobacco smoke, pesticides, microbes and allergens, and combustion products. Other factors affecting indoor air quality may also be addressed.

Investigations of each pollutant or category include: characteristics and sources of the pollutant, health effects related to or suspected from exposure to the substance, history and scope of the problem, techniques of measurement and mitigation, assessment of risk to the population, and existing approaches at the federal and state level as well as in the Commonwealth.

Data and information have been assembled from presentations by and communications with those with expertise, the scientific literature, certain secondary sources, Commission members and other interested participants.

From its inception early in the year through the end of October 1987 the Commission investigated radon and formaldehyde as elements of the overall problem. However, since study of these topics is ongoing, major recommendations cannot be made until all the subjects and issues have been presented and addressed.

INTERIM REPORT

This is an interim report addressing the many issues surrounding indoor air quality in Massachusetts. This document presents the overall indoor air quality issue (Chapter 2) as well as two specific pollutants: radon and formaldehyde (Chapters 3 and 4 respectively). The final report will additionally treat the topics of asbestos, tobacco smoke, pesticides, microbes and allergens, combustion by-products, VOC's, combinations of pollutants, policy issues and the Commission's conclusions and recommendations.

* Note: A list of acronymns and technical terms is given on page viii.

Chapter 2: INDOOR AIR POLLUTION

AIR AND POLLUTION

A thin layer of air surrounding the earth makes life as we know it possible. Naturally occurring air is a mixture of gases, solid particles, water and other vapors. The major components of "pure" air are nitrogen, oxygen, carbon dioxide, and water. Carbon dioxide and water are essential to photosynthesis, the process by which plants utilize sunlight to produce their own food. Oxygen, nitrogen and water are necessary for the respiration, growth and development of plants, animals and most other living organisms.

Other substances are also present in air as a result of both natural processes and human activities. Pollen, fungal spores, bacteria and viruses, dust particles, microscopic rock fragments, and chemical compounds such as ozone and ash occur naturally. All of these and many other chemicals may be added to air as a result of activities such as agriculture, manufacturing, power generation, and residential living.

Substances and their proportions in the air mixture vary over time and from place to place. This can be due to meteorological factors such as wind, temperature, humidity, and precipitation, and to varying geological and geographical conditions. For example, dust and other particles may fall to earth with rain, ozone increases after a thunderstorm, and pollen levels are high in the northeast in spring when the forest trees flower.

Air is regarded as polluted or contaminated when any substance or substances from either natural or human sources occurs in the mixture at levels which produce an immediate or long term negative effect on human health or the environment. Pesticides from agricultural spraying can cause health problems both as gases and when they are washed into water supplies. Radiation from serious nuclear power plant malfunctions or detonations can cause almost immediate death, radiation sickness, or lead to disease. High pollen counts produce severe discomfort in sensitive individuals.

Sulfur and nitrogen dioxides and carbon monoxide are produced by combustion processes such as forest fires, and the burning of fuel in factories, other buildings and vehicles. Sulfur and nitrogen dioxides from factory emissions produce acid rain which damages plants and increases the acidity of lakes, leading to the death of fish and other aquatic organisms. High concentrations of carbon monoxide from motor vehicle emissions are fatal to humans.

INDOOR AIR

As a result of dispersion and air movement the air inside a structure will contain all of the components of the ambient or surrounding air. Ambient air enters buildings through "ventilation", which is the process of adding air to or removing air from a space, by either natural or mechanical methods.

Ventilation of a building and entry of ambient air can occur through open doors or windows, leaks such as gaps or cracks, as well as through ventilation systems.

Indoor air may also contain substances generated by occupants or their activities, objects in the building, or the structure itself. Occupants generate airborne substances through food preparation, fuel burning for heat or cooking, tobacco smoking, hair sprays, cleaning products, paints, and the occupants' own presence, e.g., shed skin cells and hair. Contaminants may be produced by carpeting, plywood, concrete and pets. The air of an office might additionally contain chemicals from copying machines, pesticide spray residues, and air conditioning coolants. A factory may have particles and fumes from its manufacturing processes.

Since free passage of air is limited in buildings, substances emitted inside a building tend to accumulate. As a result indoor air may contain concentrations of some compounds which are greater than the ambient air. For example, an Environmental Protection Agency (EPA) study has shown that organic contaminants can occur indoors at up to 10 times the outdoor levels found in either industrial or rural areas. The level of a given substance tends to increase in winter when windows are kept shut and to be higher in structures with poor ventilation and in settings where greater amounts of a source exist.

Results of time budget analyses show that the bulk of most people's time is spent indoors. In addition, studies in the United States, Canada and Europe indicate the greatest exposure to many pollutants occurs inside buildings (1). Thus it is important to determine which substances people are being exposed to indoors, the sources of these substances, and the effects which they have on human health - individually and in combination.

INDOOR AIR POLLUTANTS: TYPES, SOURCES AND HEALTH EFFECTS

Health Effects

Indoor air pollution is a growing problem in the United States and accounts for up to 50% of all illnesses. It carries a cost estimated at 100 billion dollars per year (1). Some airborne substances are harmless; some are hazardous, i.e., have documented health effects; the effects of others are unknown. Hazardous substances identified in indoor air include: radon, formaldehyde and other VOC's, asbestos, tobacco smoke, pesticides, microbes and allergens, and combustion products.

The health effects of hazardous substances found in indoor air range from the irritating to the deadly depending on the substance, its concentration and the length of exposure. Effects may be immediate or delayed, and have short term or long term impacts. Effects may vary both among individuals, based on sensitivity, and in the same individual at different times.

Long-term health effects of indoor air pollutants include cancer, birth defects, immunological problems, nerve damage, reproductive difficulties and

developmental problems. Short term effects include pneumonia, upper respiratory infections, and allergic asthma (2,3).

Even brief exposures to elevated concentrations of some pollutants, such as formaldehyde or carbon monoxide or nitrogen oxides, can lead to headaches, dizziness, nausea, eye irritation, and respiratory problems such as wheezing, coughing, congestion or shortness of breath. These symptoms often disappear when the source is removed (4,5).

Assessment of the health impacts of indoor air pollutants may be complicated because the effects of some contaminants differ based on concentrations and/or duration of exposure. For example, a substance may have an offensive odor at low concentrations, but produce eye irritation at higher concentrations. In addition, prolonged exposure to odor decreases odor sensitivity, i.e., people become adapted to a smell, while prolonged exposure to an eye irritant aggravates it (6).

Some people are hypersensitive to a range of chemicals found in homes. They react, for example, to formaldehyde, petroleum products, cigarette smoke, and chemicals in building products like preservatives, dyes, plasticizers, and adhesives. Reactions range from mild discomfort to life threatening allergic reactions (7).

Most of the long range effects of airborne substances are known from workers who, following exposure to high levels on the job, developed a range of medical problems including respiratory diseases and cancer. Levels to which they were exposed were usually many times higher than levels to which people are exposed in buildings.

Little is known about long term, low level exposure as may occur in homes or workplaces (5). Until there is evidence to the contrary it is prudent to assume that if a substance causes problems at high levels there could be a risk at low levels, especially if exposure occurs over a long period of time.

Categories of Pollutants

Each category of pollutants and its associated health effects is described briefly below. Radon and formaldehyde are treated extensively in Chapters 3 and 4 respectively.

Radon

Radon is a radioactive gas arising from the natural decay of uranium in rock and soil. Radon gas may enter structures through pores and cracks or dissolved in well water. When radon-laden water emerges from a tap radon gas escapes into the room air. Building materials made from rock containing uranium are an additional source. Structural elements such as stone foundations, walls or fireplaces may also produce radon (8). Levels above the EPA's recommended safe level for radon have been reported in many residences in the United States (9).

Inhalation of the radioactive compounds produced as radon spontaneously decays has been associated with lung cancer in miners (5). Risk estimates of the effects of long term low level exposure are widely accepted and of concern. Studies are underway to determine: areas of high radon potential, the factors contributing to high indoor radon levels, and the extent of elevated radon levels in the United States as well as the effects of long term low level exposures.

Formaldehyde

Formaldehyde is a pungent smelling gas which causes irritation of the eyes, skin and respiratory system, and is regarded as a probable carcinogen by EPA. It has produced nasal cancer in animals (5). Major sources of formaldehyde in indoor air are bonded wood products and urea formaldehyde foam insulation (UFFI). Formaldehyde is also found in home furnishings such as carpets and draperies, and many domestic products.

Bonded wood products, such as plywood and particleboard, are widely used as paneling, flooring, in furniture and cabinets. Mobile homes have higher amounts of bonded wood products and higher formaldehyde levels than conventional homes. Homes may be grouped into 3 categories, based on method of construction: "conventional" homes which are built almost entirely on site; "mobile" homes which are provided with a chassis and transported to a site as an entity or in 2 or 3 parts; and "other manufactured" homes which include modular, paneled and kit built homes.

Elevated formaldehyde levels produced by emissions from UFFI led to a ban by the Consumer Product Safety Commission (CPSC) - which was subsequently overturned in court. UFFI has been banned in Massachusetts since 1979.

Asbestos

Products containing fibers of the mineral asbestos are widely used in acoustical and thermal insulating material including pipe insulation, and in concrete, spackling compounds, ceiling and floor tiles and ventilation systems. Over 85% of the asbestos found in buildings is immobilized within some type of binding material. Release of high concentrations of fibers occurs when such material is disturbed, either accidentally or during maintenance, renovation, or removal. Exposure to asbestos fibers has been linked to lung and gastrointestinal cancer and other diseases. For example, prolonged exposure to high levels of asbestos can lead to asbestosis, a chronic lung problem typified by shortness of breath and extensive lung fibrosis. Skin irritation may also occur (8,12).

Tobacco Smoke

Several thousand compounds have been identified in tobacco smoke including respirable particles, nicotine, polycyclic aromatic hydrocarbons, nitrogen dioxide, and acrolein (11,13). There is increasing evidence that passive exposure, i.e., exposure to other people's tobacco smoke, affects respiratory

health (11). It may cause irritation, coughing, sore throats and sneezing (14). Prolonged exposure to carbon monoxide and nitrogen dioxide may lead to chronic respiratory problems (5). An association has been found between eye irritation and carbon monoxide from the combustion of fuel or tobacco (4).

Although around 33% of adults smoke cigarettes regularly, a much higher percentage of children are routinely exposed. For example, 76% of children in a middle income community surveyed in St. Louis lived in homes with one or more smokers (15). Increased respiratory problems have been documented for infants in homes where parents smoke (16). There is evidence that passive exposure to tobacco smoke might increase the risk of lung cancer (17).

The presence of tobacco smoke may enhance the effect of both formaldehyde and radon (18). Burning tobacco produces formaldehyde which will add to concentrations from other sources, and also produces particulates to which the radioactive compounds formed when radon decays may attach and be inhaled.

Pesticides

Pesticides may be used indoors to kill roaches, ants, silverfish, wasps and hornets, and other insects and small invertebrates. Pesticides are often applied as sprays, or as liquids which evaporate and linger in the air. Vapors from outdoor applications especially on lawns or around foundations may enter indoor air. Airborne pesticides may be inhaled or settle on water or food and be ingested. Pesticides contain a variety of toxic compounds which have been identified as toxins, carcinogens or mutagens. Health problems may be associated not only with the active ingredients of pesticides but with inert compounds also contained in the products.

Microorganisms and Allergens

Inhalation of microbes, such as bacteria and viruses, which have been exhaled by other people and sometimes animals, is a primary route of transmission of most acute respiratory infections (19). Higher incidences of colds and flu during the colder parts of the year are probably the result of decreased ventilation inside buildings when windows and doors are kept shut. Serious disease and even death may result from microbes in indoor air. Tuberculosis, measles, smallpox, and staphylococci are transmitted by ventilation systems in schools and hospitals. Bacterial aerosols are incubated in toilets, ice machines, and carpets and distributed by humidifiers and cooling equipment (e.g. Legionnaires' disease) (20,21). Microbial hazards may be the most hazardous and also the most preventable category of indoor pollutants in supersealed buildings (3).

Allergens can lead to considerable acute respiratory and other discomfort, and long term exposures can cause chronic conditions. Indoor allergens include, e.g., pollen, molds, other fungi, hair, insect parts, and chemical additives. Pollen comes from cut flowers or flowering plants as well as from outside. Molds and other fungi grow on surfaces in warm, dark, damp areas, especially bathrooms and basements, and are generally worse in summer. Insects enter a structure and die in light fixtures or by windows, dehydrate and fall apart.

Insects living indoors in food areas, corners, dust, and vents, produce eggs, cocoons, webs, shed skins and feces which can enter the air. Pets and people shed hair and dead skin cells. Dust mites present in all indoor environments, feed on such debris from both humans and pets. These mites thrive in humid conditions. Their feces are an allergen and can cause asthma (22,7).

Combustion Products

The products of fuel burning for heating and cooking contain numerous hazardous compounds including formaldehyde, nitrogen and sulfur dioxides and carbon monoxide. These can accumulate indoors when stoves, heaters or furnaces are improperly vented or unvented, or when exhaust from internal combustion engines infiltrates buildings through open windows and ventilation systems. Carbon monoxide, for example, is fatal in high concentrations, and low concentrations may lead to problems such as impaired vision and brain function. Carbon monoxide interferes with the ability of the blood to carry oxygen (5).

Volatile Organic Compounds

Formaldehyde is one of many VOC's which are contaminants of indoor air. People emit acetone, alcohols, butyric and other acids. Certain (polycyclic aromatic) hydrocarbons are produced in the combustion of tobacco, wood and kerosene. Pesticides can release organophosphates or chlorinated hydrocarbons. In addition chlorinated compounds, acetone, ammonia, toluene, and benzene are found in paints, lacquers, varnishes, cleaning materials, and personal care products. An EPA study of 20 VOC's in the indoor air to which people were actually exposed along with the outdoor air, drinking water and breath of 600 residents of New Jersey, North Carolina, North Dakota and California indicated that the personal exposures to 11 of them were consistently higher than in outdoor air (10). Health risks of many of these have yet to be fully investigated (11). However, many are known or suspected human carcinogens, mutagens or teratogens. Some people (referred to as 'chemies') are extremely sensitive to fumes from household products such as aerosols, paints, cleaning products.

HISTORY AND SCOPE OF INDOOR AIR POLLUTION

History

Indoor air quality may have been an issue as long ago as the discovery of fire. Ceilings of prehistoric caves show layers of soot (14). Prior to the availability of techniques to measure substances in indoor air the only efforts at control involved dilution of unpleasant odors. This arose from a concern that smelly places must be unhealthful. In reality foul smelling substances are not necessarily dangerous, and odorless or pleasant smelling substances can be very hazardous (23).

Certain indoor air problems have been known in industrialized countries for decades. Minimum ventilation requirements began to appear in model building codes in the 1940's and 1950's. However, the widespread use of these codes did not come about until the 1970's.

A school building code has been in place in Massachusetts since the 1940's. However, Massachusetts' first comprehensive building code for new construction was adopted in 1975. Some town codes prior to this date included ventilation considerations. Others did not. Thus a sizable portion of our existing building stock has been constructed without any minimum acceptable standards of ventilation. Moreover, ventilation requirements in codes are intended to reduce the transmission of airborne respirable diseases, to control odor and humidity, to provide combustion air for heating and to remove cooking by-products. Nonetheless, past concern about air quality was directed toward outdoor and industrial settings. Government efforts at quality control were similarly concentrated in these areas. Over the past several decades some new indoor pollutants have been discovered, e.g., radon, and new concern has arisen about the health effects of others, e.g., formaldehyde, pesticide residues.

Buildings consume 1/3 of America's energy (6). When energy costs increased in the early 1970's there was a move toward decreased fuel consumption and alternative fuels. Both have led to indoor air quality problems. Increased building insulation and general tightening of construction to prevent leakage were important conservation measures initiated in response to high fuel costs. High levels of insulation were placed in ceilings, floors and walls - practices which tighten houses by blocking routes by which warm air escapes. Ducts of heating, ventilating and air conditioning (HVAC) systems were sealed and heavily insulated (24). Thermal doors and windows were developed and installed (5). Heavily insulated shades and curtains were pulled across windows at night.

A by-product of tighter buildings was a decrease in air exchange rate, i.e., the rate at which the air in a room or structure is replaced by "new" air and the concentration of pollutants is decreased. Air exchange rate is usually expressed as number of air changes per hour (ach). Large numbers of wood stoves were installed in the 1970's. Lack of regulation led to high concentrations of emission pollutants such as carbon monoxide, nitrogen dioxide, and formaldehyde when stoves were not properly vented (14). Other improperly vented heating and cooking appliances and synthetic building materials poured contaminants into indoor air. Radon and tobacco smoke accumulated. The decrease in air exchange rates meant that contaminants entering a room were being removed at a lower rate and thus reaching greater levels.

Problem Scope

The extent of indoor air quality problems is as yet unknown. Elevated radon levels have been found in structures in most states (25,26); up to 1/3 of Americans smoke cigarettes (8); formaldehyde and asbestos are still found in products in many buildings; pesticides are widely used; microbes and allergens have seldom been measured; and alternative fuels are growing in use.

The indoor air quality issue is complex. While many sources of pollution have been identified, information on levels found in structures, particularly homes, is available for only a few pollutants. Little is known about long term health effects of exposure to either single pollutants or to several simultaneously. Since indoor air may contain many different substances questions regarding health risk must be asked for each substance and for combinations of these substances (since interactions could potentially occur). That is, if both substance "A" and substance "B" are present they may react. "A" might cause more severe effects in the presence of "B" than when it occurs alone. If levels vary widely it will be difficult to predict the risk of illness to any one individual (5).

Sick Building Syndrome

In recent years people who live and work indoors have reported many symptoms of physical discomfort such as fatigue, headache, sore throat, nausea and eye irritation. Such symptoms have appeared most frequently in tightly constructed buildings, often without operable windows. This has led to the phrases sick building syndrome (SBS) and tight building syndrome (TBS) (27).

SBS has been known since World War I. The first published study was in England in 1948 (3). According to Woods, Janssen, et. al., SBS occurs when three conditions are met. First, symptoms such as those described above occur in more than 20% of the building occupants. The 20% arises from a thermal comfort ventilation rate guideline established by the American Society of Heating, Refrigeration and Air Conditioning Engineers (ASHRAE) which specifies a certain number of cubic feet of fresh air per minute (cfm) per occupant. The guideline is based on the cfm which will be comfortable for 80% of the visitors to a room (27).

Second, the cause of the problem is not recognizable (27). In some cases there is no obvious source of contaminants. In others the source is known but the means by which it is transmitted from its point of origin to the area where complaints occur is not known (28).

Third, most people with discomfort indicate relief almost immediately after leaving the building. An SBS problem is considered resolved when the cause is identified and addressed and any modifications made such that complaints cease (27).

Causes of SBS may be either physical or psychosocial. A physical cause is almost always found, for example, poor ventilation or contaminated air. The ventilation system is often the major problem either because it is not achieving an appropriate air exchange rate or the system is itself a source of contamination. An adequate air exchange rate may not occur due to: poor design or maintenance; lack of capacity - if the building has changed use or has been expanded while the system remained unaltered; or lack of use of the system (29). Some sources of contamination which have been found include: outside pollutants, e.g., carbon monoxide, nitrogen dioxide from automobile exhaust which are drawn into the building; microbes and fungi which incubate in ventilation ducts; and substances circulating from other parts of a building, e.g., fumes or smoke. Psychophysical factors such as stress or job dissatisfaction are often found in addition to a physical cause (27).

Perhaps the best known case of SBS is the one which led to the discovery of Legionnaire's disease. In 1976, 221 American Legionnaires attending a convention in Philadelphia became sick and 29 of them died. The cause of their illness was ultimately determined to be a disease associated with bacteria in the cooling towers of their hotel. The bacteria were named Legionella which, along with other harmful bacteria, have since been found in other buildings (3).

Contamination in other cases has since been traced to water towers, ventilation systems and air ducts. With poor maintenance bacteria such as Legionella, staphylococcus and streptococcus, and fungi such as Penicillium can breed and enter the air circulating in the building (29).

Numerous other occurrences of SBS are known. In 1984 two dozen employees at the New York Times became sick. The cause was found to be birds' nests and feces located near the air conditioning equipment which contaminated the air (3).

In an elementary school outside of Washington, D.C. four teachers lost a total of 350 days of work due to problems in the building. The school was built in a low area with no provision for drainage. Water leaks were found in the walls. Dampness led to mold growth on books and materials (3).

People working at the veterinary school at the University of Florida in Gainesville became sick in 1986. The cause was a faulty ventilation system. Thus far the university has spent \$1 million appropriated by the Florida legislature to purchase modular buildings to temporarily house staff. The cost of repairing the ventilation system is \$3.4 million, 1/3 the cost of constructing the building in 1978 (29).

SBS may be widespread. Fireman's Fund Insurance Company found that 1/3 of the 48 buildings they investigated around the United States posed indoor air pollution hazards (24) and subsequently, in 1983 established an SBS laboratory and consulting service (30). By 1986 the National Institute of Occupational Safety and Health (NIOSH) had investigated complaints in 450 public and

private office buildings. Problems were found with fabrics, microbes, and contaminants from both inside and outside. Fifty percent of the buildings had inadequate ventilation (31).

MEASUREMENT AND MONITORING OF INDOOR AIR QUALITY

Pollutant Identification and Concentration

In order to evaluate excessive indoor air pollution and its health effects it is important to identify which pollutants are present in a room or building, and to determine how the levels of each vary with time. Monitors are available for particulates and a few gases such as radon, formaldehyde, nitrogen dioxide, sulfur dioxide, and carbon monoxide (32). Where interest is in a specific pollutant monitors can be installed for a period of time and the results analyzed in a laboratory.

In situations such as SBS the source of contamination may be unknown, and testing for a broad spectrum of possible pollutants may be required. Testing is achieved through gas chromatography or spectroscopy. Methods are complex, time-consuming and require expertise and specialized equipment (5).

Total Individual Exposure

The effect of airborne pollutants on a given individual may be cumulative. The concentration and types of pollutants vary over time, within a room, from room to room within a building, between buildings, and from indoors to outdoors. An individual divides his or her time among many places, both indoors and out. Since the effects of pollutants often depend upon the total exposure an individual receives it is important to monitor the individual through his or her daily activities.

Methods to determine an individual's total exposure to indoor airborne pollutants may be subjective or objective and represent either an assessment of existing conditions or a reconstruction of past exposure. Determination of the amount of exposure may be via anecdote, history, activity diary, or personal monitoring.

"Anecdotal information" or recollection relies on a person's memory of past conditions and may provide valuable clues to a present physical condition. However anecdotal information is often vague and inaccurate. There may also be inaccuracies because some contaminants are colorless and odorless and some like radon decay products and microorganisms may be below perception thresholds (14).

A "history" is often more specific and may consist of medical records, and data such as the number of smokers in the home at various times, occupational background, and living conditions (e.g. over rock high in uranium).

A personal or "activity diary" is an ongoing record of daily activities, e.g., amount of time spent indoors vs. outdoors and the characteristics of the indoor environments. If this is coupled with measurement of the pollutant levels in the areas where time is spent, an exposure profile can be developed.

Because of the variability of pollutant levels over time and from place to place and, since most people do not remain in one place all the time, the only means of quantitatively assessing total exposure is "personal sampling". With this technique a person carries a monitor while going about his or her usual activities.

A number of limitations exist with personal sampling in addition to the lack of availability of monitors for many gases: analysis can be costly and time-consuming; many industries will not allow samplers on their premises so the people sampled may not be representative of the general population; monitors may be cumbersome or annoying so some persons may not wear them; and wearing them may modify behavior.

RISK ASSESSMENT FOR EXPOSURE TO INDOOR AIR POLLUTANTS

Risk Assessment is a tool developed by epidemiologists and toxicologists to determine the probability of a given health effect arising in a population from exposure to a given chemical substance or mixture of substances. A model presented to the Commission by John Graham of the Harvard School of Public Health utilizes three factors - hazard identification, dose response and exposure assessment - to develop a risk or probability profile for a substance (2,33). The following discussion is based on this model.

The first component, hazard identification, is a determination of whether the substance affects human health and, if so, in what ways. For a substance to be deemed a hazard a causal link must be found between the substance and one or more health effects.

Once the hazardous nature has been established it is important to know the likelihood that various concentrations will produce the health effect(s) and how their severity varies as concentration increases. This is dose response, the second component of risk assessment.

Exposure assessment, the third component, seeks to determine whether a hazardous substance actually occurs at health threatening levels.

Hazard Identification

A substance is hazardous if it presents a health risk, i.e., its presence leads to discomfort, disability, disease or death. Evidence to support the hazardous nature of a substance may come from structured human or animal studies, exposed occupational groups, histories including medical records, anecdotal information, or personal diaries. Each has strengths and limitations.

Studies of laboratory animals permit testing of sufficient numbers of individuals to produce statistically meaningful results. Short life spans of small animals permit lifelong and multigenerational studies yielding results within a reasonable period of time. However, results of animal tests are not always extrapolatable to humans because of metabolic differences among species and the use of test dosages which may be higher than those to which humans are exposed (34).

Occupational studies of workers exposed to a hazardous substance are often the first elucidation of the hazardous nature of a substance. However levels of the hazardous substance are not always known and other substances may also be present making it difficult to pinpoint the cause of a health problem.

Medical records may provide a profile, e.g., occupational history and lifestyle, including time spent in various environments, and personal habits such as smoking or diet. However, it is difficult to extend conclusions regarding the hazardous nature of a substance based on studies of medical records to the general population. This is because patients don't represent a random cross-section of a population but rather those individuals who have developed problems.

Anecdotes may help illuminate a problem yet it is sometimes difficult to know their accuracy. Anecdotal information may suffer from bias on the part of the patient, or forgetfulness of events or situations which occurred in the past. Personal diaries may be incomplete if an individual forgets to keep the diary or eliminates certain events or situations because they are not considered relevant or the person is embarrassed to report them.

While each individual study or type of study may leave some uncertainty as to the hazardous nature of a substance, as more studies accumulate a clearer picture emerges. The hazardous nature of substances in indoor air in the pollutant categories the commission is studying is already established.

Dose Response

The hazard posed by a substance may vary with concentration (dose) and/or with total exposure i.e., a dose over a period of time. "Dose" response studies are used to assess the health effects of different doses or total exposures to a pollutant. Such studies can indicate when a health effect appears and how its intensity varies as dose or total exposure increases. Dose response studies are generally undertaken once a substance has been shown or is strongly suspected to be a hazard and may utilize any of the methods described above.

The dose response relationship may take one of several forms. It is linear if the magnitude of a health effect increases in direct proportion to either dose or total exposure (Fig. 2-1). A sigmoid relationship occurs if at certain low doses or exposure times there is no health effect (Fig. 2-2). At some threshold an effect occurs and then increases as in the linear model. There may also be a point where increased doses or exposures no longer increase the magnitude of the effect, e.g., if a certain dose of a substance causes death, higher doses can't cause more death. In addition to linear and sigmoid curves, other relationships may also occur.

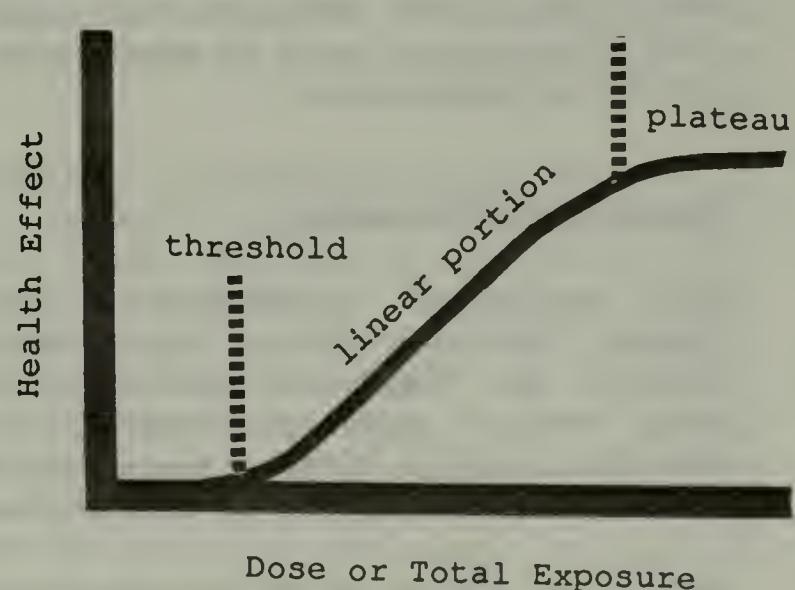
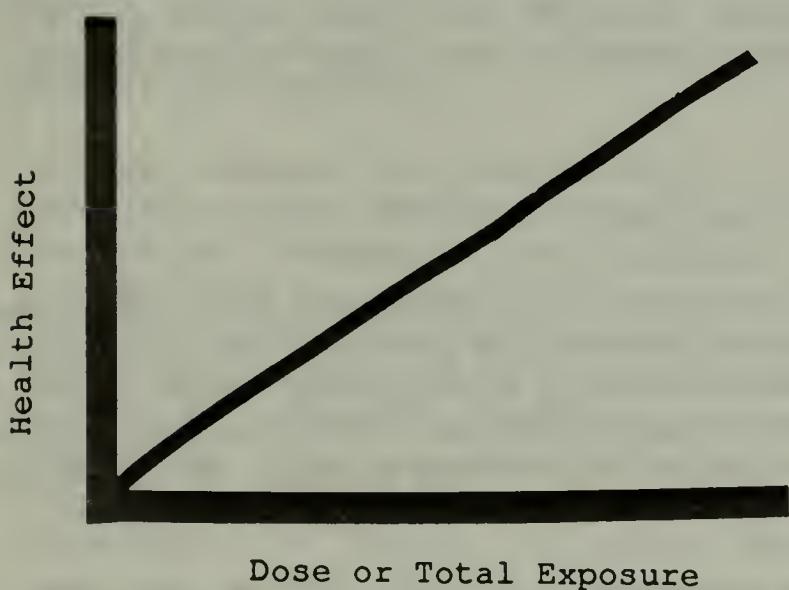


Figure 2-1. Linear Relationship of Health Effect to Dose or Total Exposure

Figure 2-2. Sigmoid Relationship of Health Effect to Dose or Total Exposure

For some substances hazardous effects are known only for high doses and studies are needed to determine the effects of lower doses, and whether a threshold exists.

Exposure Assessment

Once a substance is established as hazardous and its dose response relationship is known, exposure assessment may be used to determine whether health-threatening levels do in fact exist. To assess exposure to indoor air pollutants it is important to establish not only whether health-threatening levels occur, but also how much variation there is from structure to structure or from time to time in the same structure, how many structures are affected and which individuals are being exposed to which levels. Extensive studies may be needed to develop a full profile of exposure.

Risk Characterization

Knowledge that a substance presents a hazard, along with dose-response information and exposure assessment, allows development of a profile which characterizes the nature and magnitude of the human risk. Risk characterization is often expressed as the number of persons likely to be affected (x) per one hundred thousand population.

The scheme outlined above is a framework to assess a problem. In an ideal situation the nature of a hazard, all its possible health effects, its interactive effects with other substances, its dose-response behavior, and the full extent of the population's exposure to the problem would be known. Policy decisions, however, are often required in the absence of or uncertainties in complete data in one or more of the above categories. Factors such as extrapolation from animals to humans, whether significant numbers of structures have been assessed to extend the results to all structures, how measurements were taken, and for what time period and with what frequency, must be weighed in deciding when or what type of governmental action is appropriate.

Massachusetts Methodology

Risk assessment techniques for ambient toxics in Massachusetts were developed jointly by the Office of Research and Standards and Division of Air Quality Control in the Department of Environmental Quality Engineering (DEQE). Their goal was to develop health-based recommended ambient air levels. This Chemical Health Effects Assessment Methodology uses three factors in assessing health effects: weight of the evidence, potency, and severity of the effect

Weight of the evidence is a qualitative assessment of the strength of the data - of how convincing it is; potency is a quantitative assessment of the dose response; and severity of effect is utilized to distinguish minor effects, such as irritation, from more serious irreversible effects.

These factors are used to assess each of four categories of health effects resulting from exposure to a hazardous substance: acute/chronic toxicity,

carcinogenicity, mutagenicity, and developmental and reproductive toxicity.

The Department of Public Health (DPH) uses the four major steps outlined by the National Research Council (NRC) and discussed above in conducting risk assessments: hazard identification; dose-response assessment; exposure assessment; and risk characterization.

INDOOR AIR POLLUTION MITIGATION

There are two basic strategies which may be applied to mitigate unacceptable levels of airborne pollutants in structures - addressing the source of the pollution and addressing the levels of contaminants in the air. These may be referred to as "source control" and "removal" respectively.

Source Control

Sources of pollution can either be man-made or exist in nature. When practical, source control is the preferred mitigation procedure for minimizing the level of an airborne pollutant since it prevents or reduces the ability of the source to become an indoor contaminant. Total elimination or avoidance of a source is a permanent solution. Other control techniques will reduce contamination levels by reducing source quantities or by sealing procedures.

Source control measures are pollutant specific and there are a number of current examples. These include the use of low formaldehyde emitting construction materials or substitution of non-emitting materials. Cigarette smoking can be banned in public places or in homes, foundations sealed to reduce radon entry, insulation other than asbestos used, paints and solvents stored outside of an occupied space and safer cleaning and health care products substituted.

Another example of source control is treatment of contaminated equipment and materials with microbial solutions, e.g., medical and laboratory supplies and facilities, carpets, and furniture. The most appropriate solution to use, however, is a matter of controversy. Chlorine bleach is effective but, because it is caustic to eyes, nose and skin, caution must be used. Ammonia is not always effective. Glutar-aldehyde based products are used in hospitals, doctor's offices, laboratories and industrial facilities on everything from equipment to walls. Some of these burn the eyes and nose and have a strong, lingering odor. However more acceptable forms are being developed. A mixture of sodium chlorite and water is also used. Preventive measures against microbial contamination include maintaining water temperature above 120 F, keeping relative humidity below 70%, using steam humidifiers and avoiding standing water in HVAC systems (3). When an HVAC system is the contamination source proper maintenance may be the solution, or redesign may be required, e.g., to prevent birds from nesting in the cooling tower or to create accessibility to ducts where organisms are breeding.

Removal

Contaminants may be removed from a building and replaced with outdoor air by means of passive or active ventilation, or they may be removed from the indoor air without replacement of outdoor by air cleaning devices. If contaminants are generated at a faster rate than they are removed, the concentrations in the air will increase. Conversely, removal at a faster rate than generation will result in a decrease in concentration of those contaminants. Ventilation is effective for removal of all indoor air pollutants (35) to a greater or lesser extent depending upon the pollutant.

Passive Ventilation

Structures without active ventilation rely on open doors and windows or other intended or unintended openings in the structure for air movement in or out. Passive ventilation refers to air exchanged through these openings by natural forces. The rate at which air is passively exchanged, i.e., how well it works to remove pollutants, depends on many factors, including size and number of openings, wind speed, temperature difference between indoor and outdoor air and occupant living patterns. Generally, maximum ventilation rates will occur during extremes of wind velocity and outdoor air temperature. Negative indoor air pressures can be induced by these factors and in turn increase the rate of radon entry into homes.

Most of the 82 million homes in the United States have no mechanical ventilation and current residential building practices rely principally on passive ventilation to change the air. The energy conserving tight construction of recent years has reduced the amount of air flow and therefore the rate of pollutant removal. A typical new house in the northwestern United States experiences 0.6 ach while duplexes, apartments and mobile homes range from 0.3 to 0.6 ach. This may not always be adequate since present information indicates high levels of certain pollutants in many of these structures. In general older homes have about 1.0 ach and newer homes around 0.5 ach (31).

Active Ventilation

Active ventilation occurs when electro mechanical devices such as blowers are used to achieve an exchange of indoor and outdoor air. Active "exhaust-only" systems rely on unintended or planned openings in a structure for outdoor air supply. Typically, the most effective system of this type for ventilation of an entire structure would provide one or more points of active, continuous exhaust, connected by ductwork to remote spaces having openings for entry of outdoor air. Spot ventilation provides for intermittent removal of certain pollutants at their source, i.e., both fans and range hoods, whose effectiveness relies on openings in the structure for replacement of air being actively exhausted.

Other ventilation systems actively exhaust and actively supply fresh air to a structure. Such systems provide a higher assurance of achieving designed ventilation rates. In addition, because air is being mechanically exhausted and supplied, the counter-flowing air can be ducted through air-to-air heat exchangers for the purpose of conserving energy. In the winter, most of the heat in the exhaust stream can be transferred to the colder incoming air,

recovering up to 80% of the heat that would otherwise be lost. For example, with an indoor temperature of 70 F and outdoor temperatures of 0 F, outdoor air would enter at 56 F and indoor air would exit at 14 F. Distribution of fresh air throughout a structure can be achieved by the use of separate ductwork supplying and removing air at remote locations or by circulation through existing warm air heating or air conditioning systems.

A third method of actively ventilating is to pressurize a building with outdoor air, forcing diluted indoor air out through leaks and openings. This is the typical method employed in most commercial buildings when outdoor air is drawn directly into return air ducts through adjustable dampers, displacing indoor air which would otherwise be recirculated.

For all ventilation systems, maximum efficiency will occur when exhausting from locations of maximum contamination. When pollutants are evenly mixed throughout a space and the source rate is constant, the concentration of airborne pollutants will be inversely proportional to the ventilation rate, i.e., doubling the ventilation rate will halve the concentration. This is known as the dilution principle (Fig. 2-3). Active ventilation systems provide continuous ventilation to which passive ventilation may add but not subtract. They can also provide for occupant control to suit occupant sensitivity, variable sources and source strengths.

An existing ventilation system which is inadequate because of design flaws, poor maintenance or expanded use of a building is often associated with poor indoor air quality. Mitigation can often require redesign or implementation of maintenance procedures. In cases where the outdoor air ventilation provision of an HVAC system is simply not being used the remedy is obvious.

Effective ventilation rate is the ability of ventilating air to remove contaminants from occupied spaces. By measuring the carbon dioxide concentration in an occupied space and comparing levels with outdoor air the effectiveness of a ventilation system can be assessed. Ventilation rates are commonly determined by measuring air pressure differences with a pitot tube, or by anemometers or by tracer gas techniques. Tracer, such as sulfur hexafluoride, can be released near a suspected contamination source and tracer movement through the building can be assessed (28).

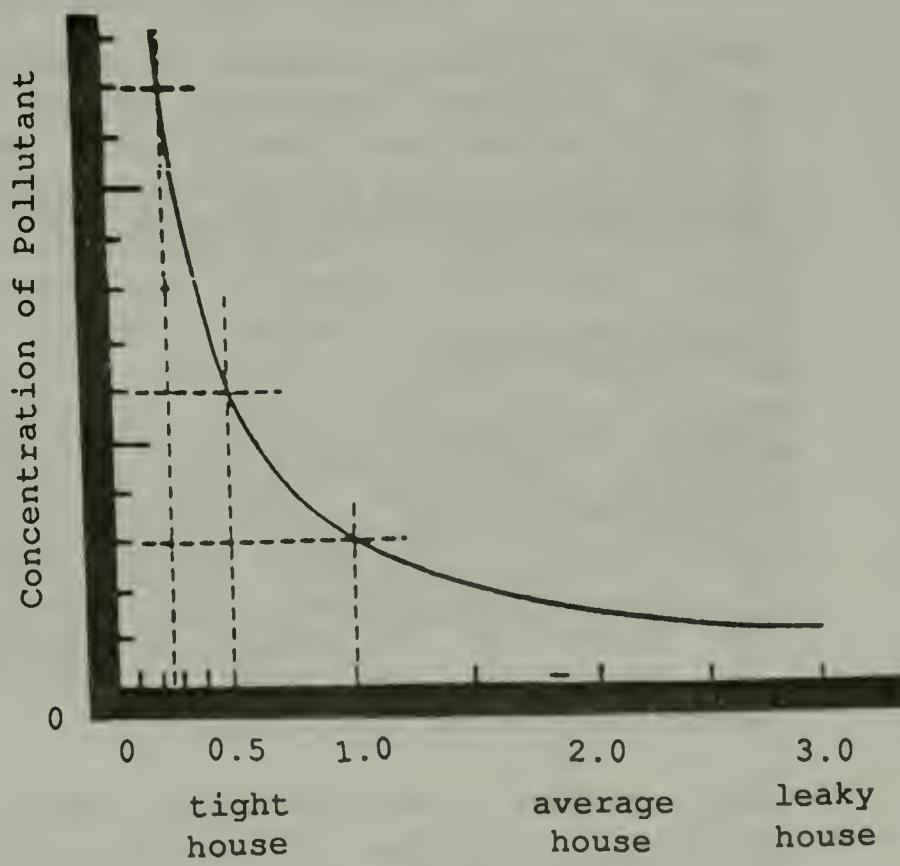


Figure 2-3. The Dilution Principle.

Concentration of Pollutant
is Inversely Proportional
to Ventilation Rate.

Air Cleaning

Various types of filtration systems which remove airborne contaminants are available. Included among these are electrostatic air cleaners, media filters, activated carbon, and air washing systems. In electrostatic precipitators (or electronic air cleaners) particles pass over electrically charged plates or wire grids, become charged, and deposit onto oppositely charged plates or grids. Media filters range in effectiveness from coarse filters such as those used in forced air furnaces to fine high efficiency particle arrester systems. Filtration is primarily applicable to particulates and not appropriate for removal of gases.

Some types of air cleaning technologies such as activated carbon and air washing systems which can remove gaseous contaminants are available. However these are generally impractical or cost prohibitive for comfort applications. Activated carbon media needs to be changed regularly and the effectiveness of electrostatic precipitators depends on regular maintenance.

INDOOR AIR POLICY

Policy Decisions

Policy provides a framework for government to address a problem such as indoor air pollution. The process of policy development involves complex interactions among affected groups with diverse viewpoints (34). A common way to build a foundation for policy is through a consensus panel which draws conclusions based on available evidence. Such a panel might include, for example, representatives from government, business or industry, and consumer groups as well as technical experts.

Spengler and Sexton present a straightforward approach for developing informed policy decisions regarding indoor air pollution. They discuss five aspects: problem definition, health risk estimation, mitigation measures assessment, identification and resolution of public policy issues, and decisions regarding appropriate government response (36).

Problem Definition

Indoor air pollution arises from a complex of contaminants from many different sources. The concentration of each pollutant in the air of a room can vary independently due to differences in the rate the pollutant is being generated and the rate it is being removed from the air. Each pollutant can lead to one or more health problems since certain combinations of pollutants may interact. The extent of the effects of combinations is unknown. Severity of an effect can depend on the level of the pollutant, the length of exposure, and the sensitivity of an individual.

Thus a clear understanding of indoor air pollution requires identification of: pollutants, sources of pollution, factors which affect the rate of pollutant entry, factors which dilute contaminants, the range of concentrations which

are found in indoor air, how exposure varies with peoples' daily activity patterns, and the types of health effects which result (36).

Until recently most indoor air quality research has focused on problem definition. Areas which have been most heavily researched include: nitrogen dioxide and carbon monoxide from unvented combustion (mostly gas stoves); formaldehyde from UFFI, building materials, and furnishings; and concentrations of airborne particles such as those present in tobacco smoke. Research is now increasing with passive tobacco smoke, radon decay products, VOC's and particulate phase organics. Although microbes may be an important source of health problems, some of which are fatal, levels of bacteria, viruses and fungi are seldom measured (36).

Risk Assessment

A complete risk profile for an indoor air pollutant would include the range of health effects which occur from the presence of a given pollutant or mixture of pollutants, how the effect differs as the level of pollutant rises, whether immediate or long term exposure is more important to the development of the health problem, what interactive effects occur among pollutants, how many people are being exposed, and the levels of exposure they are receiving, and how their susceptibility differs. When all of these are known it is possible to predict the chances of an individual developing a given health effect.

Good risk profiles are essential in order to compare control strategies to reduce risk. While data is accumulating on the above aspects of health effects it is sometimes fragmentary, anecdotal and contradictory. Dose response relationships are often unknown. Although there are still uncertainties regarding how significant indoor exposures to air pollutants are in terms of short and long term health effects, the large number of people exposed makes indoor air pollution a significant problem (36). While it is still difficult to clearly link contaminants with observable health effects mitigating measures can be used to decrease exposures and risks.

Mitigation Measures Assessment

Mitigation or relief of indoor air pollution ideally means decreasing the concentrations of pollutants in the air to levels where no long or short term health effects occur. The general types of mitigation include: source control through removal, substitution or modification of the contaminating substance; removal of contaminants through ventilation or air cleaning; and changing the living patterns of the occupants. Under current technology some techniques are more appropriate than others for a given contaminant and some contaminants are handled with a combination of methods. Behavior modification depends on awareness of a problem by the occupants or owners of a building and a willingness to alter personal or work habits. Awareness can be enhanced with public education, product testing and labeling, simple sensing devices and clarification of legal rights and responsibilities (36).

Policy Issues

A key component of informed policy decisions is a clear understanding of the

issues. Policy questions must be identified and then resolved before indoor air quality problems can be addressed. However, thus far there has been little effort to accomplish this. Policy questions include:

- (1) Does government's role differ regarding public and private structures, e.g., schools vs. homes?
- (2) Should voluntary and involuntary risks be considered separately, e.g., cigarette smoking vs. exposure to the smoke of others?
- (3) Should long term and short term exposures to indoor pollutants be addressed differently?
- (4) Should the same degree of protection be considered for the general population vs. infants, the ill, the elderly, or other particularly sensitive individuals?
- (5) How should responsibility be divided among individuals, building owners and operators, architects, developers, contractors, manufacturers and government?
- (6) Is government action appropriate and if so which type(s) should be used, e.g., regulations vs. guidelines?
- (7) At which level of government should authority and responsibility be vested?
- (8) What are the cost-benefit tradeoffs between energy conservation and indoor air quality? (36)

Scientists vary in their views of when action should be taken regarding health policies. Some advocate action based on slim evidence by appealing to emotional factors. Others won't act without solid proof from controlled experiments and often find themselves in the company of special interest groups. Some don't want to take a stand or equivocate by placing undue conditions or qualifications on their findings. They become hypercritical of all studies (34).

Government Response

While scientists and health officials recognize that contaminated indoor air is a major exposure route for many pollutants, government must decide how to protect public health while faced with incomplete and sometimes conflicting information (36).

Government responses to indoor air pollution problems might include: legislation, regulation, guidelines, suggestions, education, or no action. Possible responses include:

- (1) Encouraging research to fill data gaps central to decisions
- (2) Treating indoor air quality as a personal choice issue and emphasizing education and labeling
- (3) Developing economic incentives or disincentives to encourage adequate indoor air quality
- (4) Promoting voluntary industry codes and guidelines
- (5) Defining legal responsibilities and liabilities
- (6) Establishing guidelines to assist individuals, professionals and regulatory officials
- (7) Promulgating rules and regulations including design standards (e.g. building codes, minimum ventilation requirements), emission standards (e.g. limits for appliances, consumer products, building materials), and indoor air quality standards (e.g. maximum allowable concentrations for certain contaminants)

(8) Taking no action, based on little public health risk or unfavorable cost-benefit ratios (36).

Appropriate responses to indoor air problems depend on resolution of the policy issues, e.g., if air of private residences is considered a matter of personal choice (as was decided with saccharin or cigarettes) then the appropriate response may be education, product labeling, etc. If offices or schools are considered areas where the individual has little or no control over air quality then regulation could be appropriate (36).

Constraints on Government Action.

Federal government actions regarding indoor air pollution are currently constrained by a number of factors:

- (1) inadequate research funding
- (2) lack of firm scientific basis for action, e.g., number of people exposed, severity and pattern of exposure, health consequences
- (3) lack of consistency in approach or centralization of authority for indoor air quality issues
- (4) reluctance of federal officials to act without clear statutory authority
- (5) reluctance of regulators to become involved in controversy over whether private spaces should be regulated
- (6) fear by some regulatory and environmental people that acknowledging indoor air problems will weaken the case for outdoor standards (36).

ASHRAE

ASHRAE has been active in promoting ventilation guidelines which lead to acceptable indoor air quality. ASHRAE feels government has a role in sponsoring research on indoor air quality and its health consequences and in contaminant control technology. Its rationale is that professional societies, manufacturing or trade associations or industries lack the comprehensive interest, resources and competence to insure well-funded systematic research programs (6).

ASHRAE has made a number of proposals, including the following:

- (1) Increase in government funded health effects research. The purpose of health effects research should be to determine physiological and toxicological effects of specific contaminants. This can be achieved through: clinical exposure and animal studies, and occupational health effects research; prospective epidemiological studies following populations over time with environmental and physiological measurements; and identification of sensitive individuals and possible neurotoxic effects of specific contaminants.
- (2) National inventory of indoor air contaminants. This would identify populations at risk. The inventory should include air exchange rates, sources and resulting concentrations, and models of interactions among pollutants, air exchange rates and energy consumption.
- (3) Continuous evaluation of information. As data accumulates indoor air pollution issues should be continuously re-evaluated to consider guidelines and ventilation standards established by government in cooperation with professional societies.
- (4) Address acute problems. Government should provide education, legal, financial, and technical help immediately to deal with acute problems.

- (5) Incorporate ASHRAE guidelines into state and local codes. ASHRAE Standard 62-1981, Ventilation for Acceptable Air Quality, is suggested as the most comprehensive ventilation guideline balancing energy conservation and health.
- (6) Examine policy options to reduce indoor air pollution levels. To achieve this look at market forces, source control, manufacturing controls, property and operational regulations, and establishment and enforcement of standards.
- (7) Involve private sector. The private sector, including professional societies, should be involved in improving products, improving performance standards for instruments, materials, equipment and HVAC systems and enhancing professional practices of society members.
- (8) Assist professional societies. Professional societies should be aided in disseminating information to other professionals and the public. This can be achieved, for example, through joint sponsorship of research, publications, and conferences (6).

INDOOR AIR QUALITY: FEDERAL AND NATIONWIDE INITIATIVES

Authority

In the past concerns about air quality focused on outdoor or industrial settings and federal and state programs are concentrated in these areas (36). The EPA is the lead federal agency for air pollution control. Through the 1976 Clean Air Act Congress gave EPA authority over ambient air. Ambient, however, has been interpreted to mean outdoor air (1,36). Thus indoor air pollution is not considered to be covered by the Clean Air Act and no single federal agency is responsible for controlling it (37).

Similarly the Clean Air Act amendments of 1977 did not specifically charge EPA with indoor air quality, but it is the agency with indoor air quality closest to its central mission and is regarded as the lead agency by Congress (1). The Government Accounting Office also interprets ambient as outdoor air, but acknowledges that indoor air quality has little support because no agency has jurisdiction over non-industrial aspects and has recommended that EPA be given this authority (36).

Title IV of the 1986 Superfund Amendments and Reauthorization Act (SARA) for the first time gave the EPA a formal mandate to conduct research and disseminate information on indoor air pollution (36).

There are a number of other federal statutes under which EPA might be interpreted to have control over indoor air quality. These include:

- (1) The Toxic Substances Control Act (TSCA), which is aimed at toxic air pollutants. EPA used TSCA to require asbestos removal from schools and is considering regulatory action regarding formaldehyde exposure;
- (2) The Federal Insecticide, Fungicide and Rodenticide Act (FIFRA) applies to pesticides used indoors;
- (3) The Uranium Mill Tailings Radiation Control Act (UMTRCA) applies to tailings used for landfill in residential areas or in dwelling construction. EPA has developed guidelines for radon concentrations in homes in high risk areas;

(4) The Safe Drinking Water Act (SDWA) could be used where drinking water comes from radon-emitting soils or rocks;

(5) The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) or Superfund might be used where volatile organics or radionuclides from hazardous waste sites can travel through soil to buildings (36).

Sixteen federal agencies are responsible for various aspects of indoor air quality (37). No single federal agency has authority over the non-workplace. There is a definite precedent for authority inside buildings, e.g., the regulation of construction and operation of public buildings for health and safety reasons through building codes and fire ordinances (51). In addition to EPA those with powers over aspects of non-industrial indoor air quality are: the Occupational Safety and Health Administration (OSHA), the Department of Energy (DOE), the Bonneville Power Administration (BPA), the Consumer Product Safety Commission (CPSC), the Department of Housing and Urban Development (HUD), and the Federal Trade Commission (FTC) (36).

OSHA and NIOSH are equipped to monitor for biohazards. NIOSH, which was created as the research arm of OSHA, published guidelines for builders and building operators in the mid 1970's. These included criteria for a recommended standard for occupational exposure to carbon monoxide, a study of occupational health in hospitals focusing on indoor environmental elements, and criteria dealing with exposure to fiberglass (3). NIOSH has investigated over 450 sick buildings (31).

DOE, responsible for energy conservation programs in new buildings and residences, has funded research to improve measurement techniques and assess health effects from radon, airborne particulates and organic vapors (36). DOE has studied the relationship between indoor air quality and energy conservation and radon, including health effects, epidemiological and building science studies. EPA and DOE are developing a memorandum of understanding to coordinate their programs. DOE will have primary responsibility for basic research, with its principal focus on health effects. EPA will handle applied research, technical studies, and operational programs which deal with both the states and the private sector (1). DOE, however, lacks specific authority to address health effects, is only mandated to save energy, and can only address preventing deterioration of air quality beyond existing levels.

BPA is financing a study in Oregon to measure various pollutants in homes and the impact of weatherization techniques on indoor air quality (36).

CPSC ensures products do not create unreasonable health effects, and has banned spackling compounds with asbestos, proposed a ban on UFFI (which was overturned in court), and funded emission studies for unvented combustion appliances (36). CPSC has done work on emissions including combustion products, formaldehyde and methylene chloride (1).

HUD develops building standards for projects it funds and materials standards for mobile homes. In high radium areas in South Dakota and Montana, indoor radon concentrations must be below a certain minimum as a requirement for HUD financing. HUD has refused approval of Federal Housing Administration (FHA) loans for home construction on reclaimed Florida phosphate lands because of high radon potential and has developed regulations limiting formaldehyde emissions from plywood and particle board (36).

The FTC ensures the truth, accuracy and usefulness of consumer advertising, and recently charged two room air cleaner makers, who claimed their products remove pollutants including tobacco smoke, with false advertising (36).

The Department of Health and Human Services (DHHS) also has responsibilities for indoor air quality and regards EPA as the lead agency for indoor air quality. DHHS principally funds and coordinates a Harvard six cities study of health effects from both indoor and outdoor pollutants (1).

DOE, CPSC, DHHS and EPA are represented on the interagency Committee on Indoor Air Quality (CIAQ) and conduct, support and administer indoor air quality research (1). Currently the Committee is assessing federal indoor air research programs. Though funding will probably continue to be poor, more information about indoor air hazards will continue to accumulate, and public awareness will probably increase, ultimately leading to legislative action (36).

EPA Activities

After a presidential veto of a similar bill \$2 million was appropriated in 1984 and again in 1985 for EPA research on indoor air quality - about 3% of its overall air budget (36). Title IV of the 1986 Superfund Amendments and Reauthorization Act (SARA) for the first time gave EPA a formal mandate to conduct research and disseminate information on indoor air pollution. EPA's initial work under SARA has concentrated on the relationship of indoor versus outdoor levels of criteria pollutants (1).

In a report to Congress required under Title IV of SARA, EPA announced its intention to take a dual approach to indoor air pollution by concentrating both on individual products and pollutants and on the structures where they are found. Its program emphasizes identification, characterization and ranking of indoor air pollutants and their health effects, along with assessment and implementation of mitigation strategies. It will identify high risk pollutants and determine exposures and health risks of various populations. It will also use a generic approach looking at total exposure to indoor air pollutants and development of mitigation techniques which handle multiple pollutants using building design and management techniques (1). EPA will regard this as a buildings problem, related to the way buildings are designed, operated and used, and will use a test house to study emission factors (38).

EPA will assess federal risk reduction mechanisms and take action using existing statutes such as SDWA, TSCA, and FIFRA or through other agencies with regulatory power from Congress. It will emphasize information dissemination and ultimately increase the abilities of state and local governments and the private sector to identify and solve immediate health problems and reduce risks (1). EPA will also encourage research projects to improve sampling techniques (38).

EPA has addressed a number of indoor chemicals through TSCA and FIFRA. The statutes allow EPA to obtain information from manufacturers and processors when there is a likelihood the substance presents unreasonable risk to health or the environment. This is based on risk-benefit analysis (1).

EPA has issued the Asbestos Worker Protection Rule which protects public employees not covered by OSHA from asbestos exposure during abatement projects and has proposed an Asbestos Ban and Phase Down Rule to reduce future uses and exposures. The Asbestos Hazard Emergency Response Act (AHERA) passed in 1986 requires schools to inspect, prepare management plans and take action if friable asbestos is present (1).

EPA banned pentachlorophenol as a preservative on log-homes in 1986, and both pentachlorophenol and creosote as indoor preservatives for most uses. Phaseout of lindane in residences was issued in 1983, with total prohibition in 1986. It is currently assessing chlordane, heptachlor, and aldrin/dieldrin (1). Risk assessment results for formaldehyde were released in 1987.

EPA's air research program includes an approach called Total Exposure Assessment Methodology which uses personal and ambient monitors to determine human exposure. VOC's for example were found to exceed outdoor levels by up to 500%. Via the Integrated Air Cancer Program EPA developed instruments and methods to characterize airborne carcinogens. Studies include effects on children of smoking parents (1).

EPA has built a testing chamber to study pollutant emissions of building materials and consumer products and thus far has analyzed paints, floor waxes, and other substances. EPA is currently working on standardization of emissions testing procedures and developing a model to estimate exposure from volatilization of chemicals in tap water. EPA has set water emission standards under the SDWA (39) and is considering using this statute to establish maximum levels for VOC'S since they vaporize in hot water (1).

EPA participated in a study with the National Academy of Sciences (NAS) leading to the surgeon general's report on environmental tobacco smoke and health (1).

Standards, Regulations and Guidelines

Outdoor world-wide air quality standards do not consider indoor exposures. From the 1940's to 1970's indoor exposures were based on ambient air and emissions (39). Little attention was given to indoor air except in industrial settings because it was assumed that buildings sheltered people from pollutants (40). It is now known that levels in houses sometimes exceed outdoor standards. These standards cannot be readily applied to indoor levels since people are exposed over longer periods indoors. Outdoor standards also do not always take into account the heightened susceptibility of the very young, ill or elderly (35).

EPA is responsible for establishing and enforcing National Ambient Air Quality Standards (NAAQS) (36). Through power granted by the federal Clean Air Act EPA has established standards for a few pollutants in the outdoor air designed to "protect the public health...with an adequate margin of safety" (42). Standards define the concentration of a pollutant to which most people can be exposed for a given period of time without adverse health effects. Standard concentrations are normally listed in either parts per million (ppm) of air or micrograms per cubic meter of air (5).

A number of states and organizations have developed standards or guidelines for indoor pollution levels. These apply mostly to workplace or public buildings. As yet there are no air quality standards which apply to all residences in the United States (5).

In the United States the tendency is to address comfort rather than health. OSHA regulates concentrations of pollutants in the workplace (43) and enforces industrial "consensus" standards designed to protect employees from "material impairment of health or function" (36). ASHRAE publishes ventilation system design and operational guidelines as well as minimum outside air requirements.

In 1977 ASHRAE published indoor ventilation guidelines for buildings constructed in the eight preceding years. In 1981 these were updated. Its Ventilation for Acceptable Indoor Air Quality Guideline requires higher air exchange rates in areas where tobacco smoking is allowed (3).

In public, commercial and office buildings the current ASHRAE guidelines are 5 cfm/person of air in non-smoking areas and 25 cfm/person in smoking areas. These guidelines fail to recognize pollution from newer materials and equipment, however, and are being revised to most likely 15 cfm for non-smoking and 60 cfm for smoking areas (31). OSHA is promulgating new indoor air quality standards based largely on NIOSH and ASHRAE research (3).

ASHRAE has recommended as a guideline for residential, office or retail spaces that the exposure of the general public should not exceed 1/10 of the industrial occupational standard for a given pollutant (43). The recommendation was intended to provide a comfortable environment. ASHRAE points out that this recommendation is no guarantee of protecting health, particularly for sensitive or sensitizable persons. Following the recommendation prevents short term effects for many pollutants but does not address the effects of long term low level exposure (44).

A variety of regulatory agencies in other countries as well as federal, state, and local governments have required ventilation rates on the order of 0.5 ach in dwellings. Europe and Canada are far ahead of the United States in addressing indoor air problems. The Canadian Standards Association requires 0.5 ach of mechanical ventilation in all mobile homes. The National Building Code of Canada now calls for 0.5 ach of mechanical ventilation in all new dwelling units. Canada's R-2000 Super Energy Efficient Homes Program requires a mechanical ventilation capability of 0.5 ach. Sweden and France have adopted 0.5 ach of mechanical ventilation requirements for all new construction. The California Energy Commission requires 0.7 ach of mechanical ventilation for the tightest of California's new residential buildings. Others, including South Dakota, Wisconsin, and the Northwest Power Planning Council, have adopted or are proposing requirements for mechanical ventilation which, combined with the assumed natural infiltration rate, provide 0.5 ach in residences (45).

Congress passed the Northwest Regional Power Act in 1980 directing the four northwest states to develop Model Conservation Standards in order to capture all the energy savings "cost-effective to the region" and "economically feasible" for the consumer. The Model Conservation Standards require 0.6 ach. Tight energy-efficient homes require air to air heat exchangers to achieve this (46).

INDOOR AIR QUALITY: STATE, MASSACHUSETTS AND LOCAL INITIATIVES

Other States

There has been some activity at the state and local level to cope with indoor air quality problems. Health departments receive most of the complaints and calls for information. Thirty-two states including Massachusetts have a person or program responsible for evaluating exposures to at least one indoor pollutant; 29 have some non-industrial formaldehyde assessment program. Many state and local governments have anti-smoking ordinances to reduce exposure to passive tobacco smoke (36).

California, the only state with a comprehensive indoor air quality program, has banned the sale and use of unvented combustion space heaters in dwellings (36). California OSHA has mandated owners of structures in which people work to operate ventilation systems during all working hours and operate and maintain such systems to local building standards. Owners must also keep precise maintenance and inspection records. California OSHA took this step after receiving 350 reports a year from office workers with problems ranging from headaches to severe allergic reactions (30).

In 1987 the Minnesota Department of Health began formulating a protocol for determining "whole" indoor air conditioning of a building rather than addressing individual pollutants (46). Minnesota along with Wisconsin, has developed formaldehyde standards for new mobile homes (36) and a law in effect for 11 years restricts smoking in a variety of indoor places including restaurants, retail stores, and offices.

At least 12 states require non-smoking areas in restaurants. In 1986 Rhode Island passed a statute requiring employers to make reasonable accommodation for non-smoking employees especially those particularly sensitive to tobacco smoke. This could be achieved by partitioning or ventilation but the employer was not obligated to spend money to do so (47).

Massachusetts

In Massachusetts indoor air policies and programs are fragmentary and scattered among various agencies, similar to the situation at the federal level. Presently Massachusetts has programs in the DEQE, DPH, and the Department of Labor and Industries (DLI) to address certain aspects of radon, asbestos and formaldehyde. DEQE's Division of Air Quality can control some indoor pollutants when the source occurs outdoors. DLI has issued guidelines for investigation of "bad air" complaints. The protocol includes checking for chemical exposure sources, reviewing the symptoms of the occupants, testing for certain compounds including formaldehyde and carbon monoxide. Criteria are recommended by the Division of Occupational Hygiene for ach and percent of fresh air.

Since the energy crisis of the mid 1970's DPH has received numerous complaints of poor indoor air quality, particularly in schools and buildings occupied by the general public.

The ventilation requirements in the Massachusetts Building Code for schools had been reduced from 10 cfm to 5 cfm per occupant. The reduced fresh air ventilation requirement resulted in increased respiratory infections and frequent complaints of discomfort. DPH was successful in revising the code in late 1986 to again require 10 cfm fresh outside air per occupant as was previously required.

In 1979, DPH banned the further use of UFFI in Massachusetts after corroborating numerous homeowner complaints of health effects attributable to formaldehyde exposure. A residential formaldehyde action level of 0.1 ppm was established. An air testing and evaluation program was implemented in 1986 to provide remedial relief to homeowners with formaldehyde levels above 0.1 ppm or with documented formaldehyde related health problems. Approximately 7,500 homes in the state are thought to have been insulated with UFFI; 3,000 have been air tested to determine present indoor formaldehyde levels. The average level to date in a UFFI home is 0.04 ppm with 2-3 % of the homes tested indicating levels above 0.1 ppm.

A broad 1987 statute restricts smoking to designated areas in restaurants with a seating capacity of 75 or more. No barriers, however, need be provided. In addition public institutions of higher learning must provide a certain number of dormitory rooms for non-smokers. Nursing homes must provide non-smoking sections in common areas and employees may not smoke in patient care areas. In addition no smoking is permitted in courthouses, schools, colleges, museums, libraries, trains, airplanes or airport waiting areas, the waiting areas of certain health care facilities, or day care centers except in designated areas. However, designated areas need only be provided if there is sufficient space for non-smokers.

There are local regulations in over 50 Massachusetts communities requiring non-smoking areas in restaurants. A 1981 DPH poll showed 66% of residents, including 52% of the smokers, favor restrictions on smoking in restaurants. A 1983 Gallup poll indicated 91% of non-smokers and 86% of smokers prefer either separate sections or a total ban on smoking in restaurants.

Homeowners with concerns or problems regarding indoor air quality should contact DPH or their local Board of Health (BOH).

Chapter 3: RADON

THE ELEMENT RADON

Radon is a chemical element which occurs as a colorless, odorless gas. While it does not react to form compounds, radon is radioactive. All radioactive elements undergo spontaneous decay which produces both new elements and radiation in the form of alpha or beta particles, and/or gamma rays. If the elements produced are also radioactive, further decay will occur until stable, non-radioactive elements are formed (48,49).

Radon 222 is part of a chain created as uranium 238 decays, and is the immediate decay product of radium (Fig. 3-1). Radon in turn undergoes decay to isotopes of polonium, bismuth and lead which are referred to as "radon progeny" (Fig. 3-2) (50). Decay of progeny continues until stable lead 206 is formed.

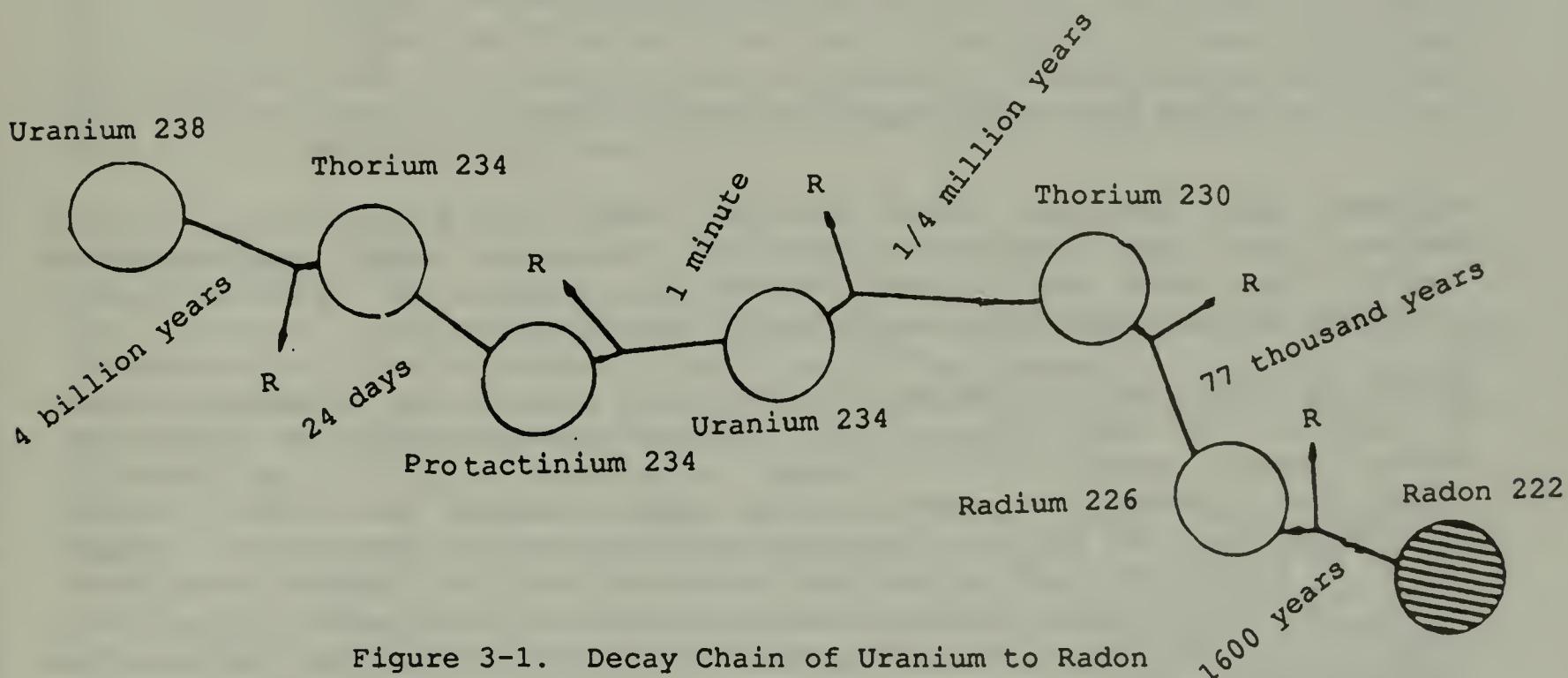


Figure 3-1. Decay Chain of Uranium to Radon

Each radioactive element decays at a specific rate called its half-life (the time required for 1/2 of the atoms in a sample to decay). Radon produced today is from the uranium present when the earth was formed. The half-life of uranium is very long. Radon, however, decays with a half-life of 3.8 days and its progeny in a matter of minutes or seconds (Fig. 3-2). Thus radon gas and radon progeny are sources of imminent radiation (50).

Uranium and other radon precursors as well as radon itself are almost ubiquitous components of rocks and soil in the earth's crust (9). Uranium is 40 times as common as gold and almost as abundant as tin (39). Radon gas escapes to the atmosphere from uranium-bearing surface rock and the soil formed as such rock decomposes. Radon from subsurface rock may also reach the surface as a gas via fissures in the rock or dissolved in groundwater.

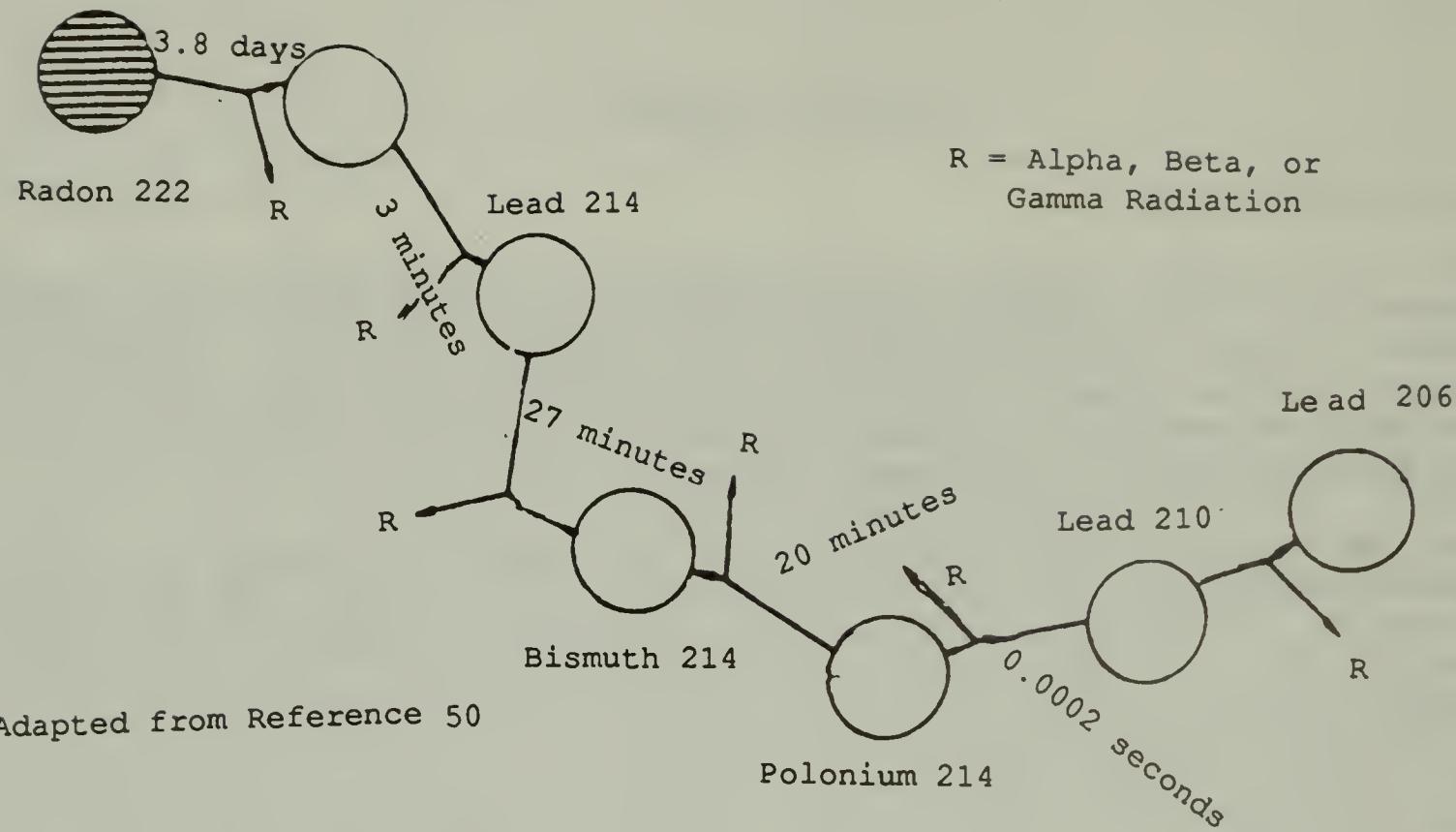


Figure 3-2. Decay Chain of Radon to Lead

Although they represent minor sources, fossil fuels such as oil, coal and natural gas may contain uranium and therefore radon. Activities or technologies which redistribute uranium, e.g., mining, well drilling, burning of fossil fuels, bring radon to the earth's surface.

Radon in coal can be released through mining and burning, in disposal areas, in runoff from fly ash, or from construction materials made of fly ash. Higher levels appear in coal mined in the western U.S. If gas production sites are located near radon-bearing rock, natural gas used in unvented appliances such as kitchen stoves and space heaters may emit radon. Propane, which is a by-product of natural gas processing, may also produce radon (48). Radon is also found in the by-products of phosphate and uranium mining and in construction materials such as brick or concrete, if they are made from uranium-bearing rock.

RADON AND PUBLIC HEALTH

Physiological Effects

While radon has no known immediate effects (51), exposure may increase risk of lung (49) cancer and is a suspected cause of stomach cancer (50). Decay of both radon and its chemically active progeny may produce damaging physiological effects in humans. Airborne radon and its progeny may contact the skin and, if inhaled, the nose, mouth, throat, esophagus, and lungs. Similarly, radon in water may contact the skin and decay or, if ingested, decay in the mouth, stomach, and intestines. Radon can pass through the wall

of the gut into the blood where its progeny can damage the stomach and other organs. Progeny, however, cannot pass through the gut.

Inhaled radiation can hit and damage molecules in living cells (50). Beta particles and gamma radiation tend to break molecules in only one place if at all. Alpha particles (which are actually fast moving helium nuclei) are the major source of health risk since they tend to cause more than 1 break in molecules they hit (48). Cells possess certain chemicals which can repair breaks. If a molecule has several breaks, i.e., is broken into more than 2 pieces, there is a chance the pieces may be put back together wrong. If this occurs with a genetic molecule (DNA) it may reproduce itself incorrectly leading to a mutation (50). Many mutations are harmful and may cause the cells containing the DNA to function abnormally. Cells may also be killed.

In the case of radon, which is principally inhaled, the major concern is from radiation affecting the function of cells in the lungs. Radon progeny are radioactive solids which will "stick to" anything they come in contact with including airborne particulates. A fraction of progeny do become attached. Both attached and unattached progeny may decay further, releasing radiation in the process (50). Radon progeny attached to particles and those existing as free ions or atoms have different likelihood of reaching the lungs and deposit in different parts of the respiratory system (48) including the lungs (49). The attached fraction is mostly deposited in the pulmonary region of the lungs with little removed by the nose (52).

Radon and Lung Cancer

The illness contracted in the late 1800's by miners in Germany and Czechoslovakia was ultimately identified as lung cancer. Mortality was 75%, and was greater for miners than for the masons and carpenters working in the same mines. In 1924 radon was suggested as the cause (50).

Uranium and other miners exposed to high radon levels have significantly higher lung cancer rates than the general population. For example, of 4000 miners who worked in the 1940's and 50's, there were 151 lung cancer deaths compared to the 16 which were expected in a comparable number of members of the general population (50). This increased lung cancer in miners eventually led to air quality standards and remedial measures in the mines.

Concentration and Total Exposure

The decay rate of radioactive material is measured in Curies (named for the discoverers of radium - Marie and Pierre Curie). A Curie is the amount of any nuclide which undergoes 3.7×10^{10} radioactive disintegrations per second. A pico Curie is one-trillionth of a Curie (5). Radon concentration (or dose) is often measured in pico Curies per liter (pCi/l). 1 pCi represents decay of approximately 2 radon atoms per minute in a liter of air (51).

Radon progeny concentration is expressed in working levels. 1 working level (WL) was originally used to define the highest level of radon permitted in a

workplace. Since alpha particles are the major cause of tissue damage, the definition of WL was subsequently adjusted to mean the amount of alpha energy potentially available in one liter of air for any combination of radon decay products. 1 WL = 1.3×10 million electron volts of potential alpha energy per liter of air. It is estimated that 1 pCi/l of radon will produce a progeny concentration of 0.005 WL (51) under ideal conditions. One working level month (WLM) is the exposure for 1 month (or 170 hours or approximately 21 8-hour working days) to 1 WL (9).

A "safe" annual exposure was originally set at 12 WLM then reduced to 3. It should be noted that WLM is defined for a workplace. In speaking of WLM's for residences it is necessary to adjust for the actual number of hours per day a person is spending in the home, which is about four times as long as a miner works.

Prior to radon reducing control measures the air of mines averaged 4000 pCi/l. Areas away from the mines measured only around 1 pCi/l. Lung cancer thus came to be regarded as an occupational problem.

Mortality rates of the miners indicate a delay of 10 to 25 years between exposure and appearance of lung cancer (50). The rate of death in the miners was found to be proportional to the total radiation exposure (radiation level \times length of exposure) (13). This suggests that total exposure may be an important variable in risk calculations.

While levels found in buildings are usually (though not always) much lower than in mines total exposures for people living or working in situations with even low radon levels can be considerable. Thus the identification of elevated levels of radon in buildings has raised concerns that such levels may increase risk of lung cancer.

RADON IN STRUCTURES: EARLY FINDINGS

Concern over radon inside structures arose in the late 1960's when elevated levels were found in Colorado homes built on uranium mine wastes. Subsequently high indoor radon levels were found in various parts of the country in buildings situated over debris from other industrial activities. Still later high levels of radon were found in homes built over natural deposits of uranium-bearing rock or soil (49).

Colorado Mine Tailings

The mill tailings left when uranium ore is extracted from rock remain high in uranium. From 1953 to the mid-1960's the use of uranium mill tailings for construction was common in Colorado - for landfill and backfill and in cement. This practice ended when the radon levels in these buildings were found to be higher than in structures not built on tailings (48).

Florida Phosphate Lands

Phosphate rock contains uranium, thorium and radium which are distributed with the products, by-products and wastes of phosphate mining. Phosphate used as fertilizer, phosphoric acid and for elemental phosphorous may therefore contain radon (1). Phosphate containing radon which is used for crop fertilizers may be taken up by plants, be deposited on the soil, or run off and enter ground and surface waters, as well as expose agricultural workers. Phosphate was once used in the United States for wallboard and plaster. Waste gypsum (a phosphate by-product) is made into concrete blocks (48).

EPA initiated studies of reclaimed phosphate mines in Polk County Central Florida in 1975. Eighty percent of phosphate mining in the United States occurs as strip mining in central Florida. After an area was strip mined the land was reclaimed by mixing mining by-products with the overburden (material above the phosphate layer) from an adjacent mine plot. About 25,000 reclaimed acres existed in the area in 1975, with another 75,000 acres potentially becoming available within the following 30 years. There may be in addition a large area where no mining is planned where uranium-containing phosphate occurs near the ground surface (53).

Thirty percent of the reclaimed land contained residential structures, 8% commercial structures and the remaining was agricultural and park land in 1975. The soil levels of radium 226, which decays to radon, ranged from 1-50 pCi/gram. Readings were highly variable over short distances. WL's of 0.015 - 0.018 (30-36 pCi/l) were found in single family houses on mineralized and reclaimed soils compared to 0.003 WL on non-mineralized soils (53).

Other Findings

When the New Jersey State Department of Environmental Protection, at federal request, surveyed sites where hazardous materials might be found, 243 homes in 3 communities were found to have been built on contaminated soil believed to be from a former radium processing plant (51,54).

In 1979 the University of California at Berkeley found radon levels of 20 pCi/l in an energy efficient house in Maryland. This exposure is equivalent to that of a Three Mile Island accident in the neighborhood once a week or the maximum exposure allowed to radiation workers. Twenty pCi/l was among highest readings ever found aside from structures built on radon contaminated industrial mine tailings.

Then in 1984 a worker at the Limerick nuclear plant in Pennsylvania kept setting off radiation alarms. His home was found to contain 2000 pCi/l of radon. Subsequent investigation showed that his house was built on the Reading Prong, a large geological formation extending into New York and New Jersey, and contained some of the highest radon levels in the country. These levels are more significant than releases from any nuclear plant and have led to widespread interest in radon in indoor air (55).

The Maryland house and subsequent radon findings on the Reading Prong were the first indications of radon in structures built on undisturbed substrate.

RADON MEASUREMENT AND MONITORING

Findings of elevated radon levels in buildings on both natural rock and soil and on ground disturbed by industrial activities prompted questions regarding the distribution of uranium laden rock and soil, whether factors in addition to uranium content of the substrate affect indoor radon levels, by which pathways enters a structure, and how widespread the radon problem might be. Answers to such questions depend in part upon accurate measurement of indoor radon levels. Since radon is colorless and odorless, and doesn't burn or glow, it is undetectable by the senses. Thus its presence can only be detected with monitoring devices.

Some monitors measure the concentration of radon gas. Other monitors measure radon progeny concentration, expressed as WL (49). There are 3 types of radon monitors: prompt, time integrating and continuous readout. Some commonly used monitors are described below.

Prompt Sampling Monitors

Prompt or grab sampling monitors are utilized to give "instantaneous" readings, i.e., of the amount of radon in the air at any given time. Detectors usually are left in place for just 5-10 minutes (51,56).

The Kuznetz method of grab sampling was developed for use in mines. An air sample is drawn through a filter which collects air particles with attached radon progeny. The filter is then aged for about an hour and the sample counted with a radiation detector. Progeny concentration is determined from detector count rate via a calibration table (51).

Time Integrating Monitors

Time integrating monitors are left in place for a period of weeks or months, where they continuously absorb radon. The total amount of radon is recorded in some fashion and calculated often through laboratory analysis. If this total is divided by the length of time the detector was left in place, the average radon concentration during the period of monitoring is determined. Charcoal canisters and alpha track detectors are commonly used time integrating monitors. A charcoal canister detector consists simply of a container of charcoal with holes in one end which is left in place for 3-7 days (5,49).

Alpha track detectors contain a piece of plastic on which alpha radiation from decaying radon leaves microscopic marks or "tracks". After exposure the plastic is developed and the tracks counted. The density of tracks is then converted to radon concentration via calibration and described as pCi/l for the time of exposure (5,49,51,56).

Continuous Readout Monitors

Continuous readout monitors sample the air and register readings continuously,

and can be used to assess the variability in radon level over time, e.g., hourly or daily. Continuous monitors can be used to provide both grab samples and integrated readings depending upon the measurement period.

Testing Protocol

The above instruments, if properly calibrated, measure accurately and precisely. However climatic and meteorological factors temperature and air pressure vary daily and annually and indoor radon levels vary accordingly. In fact levels may vary by a factor of 10 over a day or two. Thus longer term measurements than a few minutes are desirable (56). Of these the time integrating monitors are simpler to use and less costly. The two commercially, most popular and preferred radon detectors of this type are charcoal canisters and alpha track detectors.

EPA Screening and Followup Procedures

A three step radon detection procedure is recommended by EPA: a screening measurement, determination of need for further measurements based on the initial reading, lastly followup measurements. The screening measurement should be made under standardized conditions so measurements from different buildings can be compared. Testing should be done at the lowest livable level of a structure, a basement if it exists. Measurements should be made when all windows have been closed for 12 hours and the outdoor temperature is below 40 F. EPA's recommendations for subsequent action depend upon the initial results (Table 3-1). If the screening measurement is less than 4 pCi/l, the

Table 3-1. EPA Recommended Response to Indoor Radon Levels

Radon Concentration pCi/l	Radon Progeny Concentration WL	Response
<4	0.02	followup probably not required
4-20	0.02-0.1	followup with detector in place for 1 year or 1 week each season
21-200	0.1 -1.0	followup with detector in place for 3 months
>200	>1.0	immediate followup with detectors in place no more than 1 week and action taken to decrease radon level

(49)

EPA action level, followup is probably not required but it should be kept in mind that single measurements are always subject to some error. Above 4 pCi/l subsequent procedures depend on the screening level. All involve further measurement with remedial action recommended at higher levels. Followup readings should be made in each living level (49).

Radon in Water

To analyze radon levels in well water (57) a water sample is collected from below the surface with a hypodermic needle, and the sealed sample is counted with a scintillation counter.

FACTORS AFFECTING RADON LEVELS IN STRUCTURES

Factors which determine indoor radon levels include: the uranium content of local rock and soil, other soil characteristics particularly permeability, pathways into the building, ventilation and the pressure differential between the structure and the environment, and the habits and activities of the occupants (9,48,53). It is presently difficult to predict how these variables will interact to affect radon levels in any given structure.

Uranium Content of Underlying Rock and Soil

The amount of uranium present in the substrate beneath a building is clearly a major factor in the concentration of indoor radon. Some of the highest levels found occur in areas which have high levels of uranium or other radon precursors in the native soil or rock.

Most rock and soil, and granite in particular, contain 1-4 ppm of uranium. Some phosphate strata in Florida reach 120 ppm (9). Acidic rock, e.g., granite, often has higher concentrations, whereas less acidic rock, such as limestone or sandstone usually has lower.

EPA has mapped the distribution of uranium-rich soils. Such soils occur: throughout much of New England; in the Reading Prong area of eastern Pennsylvania, northwestern New Jersey, and southern New York; along the eastern slopes of the Appalachians; in the phosphate rich soils of Florida; along the Georgia and Carolina coasts; and in other areas scattered throughout Wisconsin, Minnesota and west of the Rockies (58).

The U.S. Department of Energy's National Uranium Resource Evaluation Program has used airborne detectors to monitor gamma ray production from bismuth 214, a radon decay product, in order to assess the country's uranium resources. Monitoring bismuth will give an indication of the amount of radon which is being generated (55).

A joint radon survey by the Connecticut Departments of Health Services and Environmental Protection found that geological formation was a significant

predictor of radon levels in both indoor water and air. Homes over granite formations had significantly higher radon levels than the rest of the state and homes over sedimentary formations were significantly lower. The depth of overburden correlated positively with radon in water, i.e., water coming from deeper strata had higher levels of radon (58).

In late 1986 the New Jersey Department of Environmental Protection commissioned a study of radon in residences in an attempt to relate potential radon problems with geological features. Areas believed from earlier evidence to be radon prone were more heavily sampled. Averages of 8.1 and 8.2 pCi/l were found in the 2 most northerly areas, including the uranium rich Reading Prong, whereas the statewide average was 5.4 pCi/l.

EPA's Generalized Bedrock Geologic Map of New England (Fig. 3-3), which emphasizes uranium endowment, indicates numerous regions of Massachusetts are underlain by granitic rock types with moderate to high uranium content, e.g., much of southeastern Massachusetts and Cape Cod and Cape Ann. About 20% of the state is underlain by gneisses which vary from low to high in uranium content (59).

Because of variation in uranium content and other soil characteristics radon content can be highly variable over short distances. This was found in the reclaimed phosphate lands (53) and in a study of rowhouses in Ontario, those with the highest level were adjacent to those with the lowest (50).

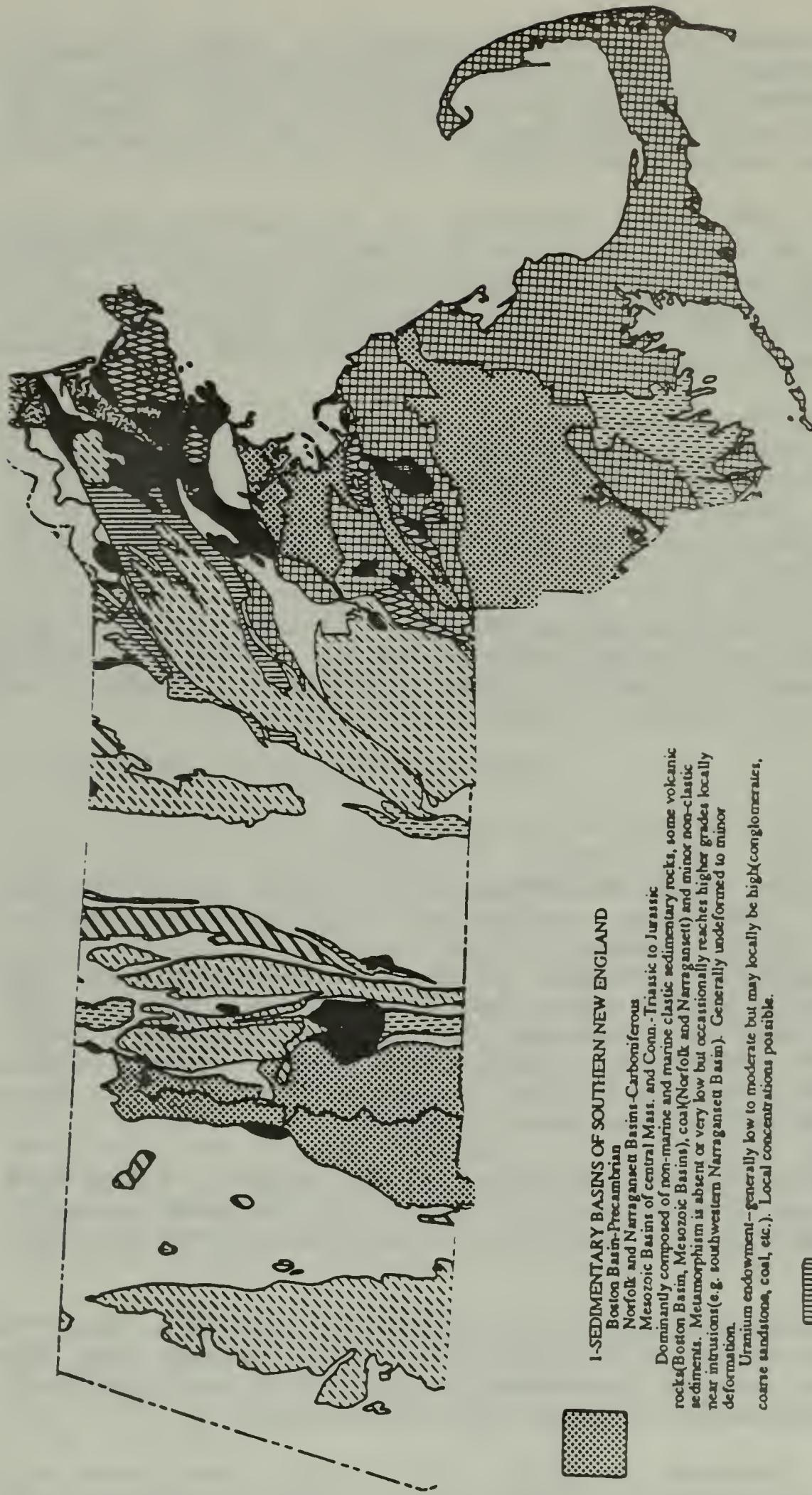
Other Soil Characteristics

Radon levels in a given soil are determined in major part by the soil composition, porosity, temperature and moisture, and also by the radon gas pressure in the soil, and the pressure differentials between the soil and air. The rate of radon diffusion from soil to the air is affected by these same variables. Diffusion is reduced when the ground is cold or frozen, wet following heavy rain, under snow cover, and when barometric pressure is high. Review of nearly 1000 measurements indicates the average worldwide outdoor diffusion rate of radon from soil is 0.42 pCi/meter square/second. The range is from 0.006 to 1.4 pCi/meter square/second (9,48,51).

Concentrations of radon in outdoor air average about 0.1 pCi/l or 0.0006 WL in the northern hemisphere. Concentrations vary with precipitation, barometric pressure, and time of day, in addition to local presence of uranium, soil structure and land use (51).

Radon reaches the atmosphere when it is emitted by soil, rock or water. A fraction of this may return to the ground. Rain may absorb some radon from the air as it passes through the troposphere where the highest atmospheric concentration occurs, and return it to soil and surface water.

Soil permeability is important to indoor radon levels. Radon formed in a bedrock containing high amounts of uranium may not reach the surface if the overlying soil is a relatively non-porous clay. Alternatively a rock quite low in uranium might lead to high indoor levels if overlain by a porous sand.



1-SEDIMENTARY BASINS OF SOUTHERN NEW ENGLAND

Boston Basin-Precambrian
Norfolk and Narragansett Basins Carboniferous

Mesozoic Basins of central Mass. and Conn. Triassic to Jurassic
Dominantly composed of non-marine and marine clastic sedimentary rocks, some volcanic rocks (Boston Basin, Mesozoic Basins), coal (Norfolk and Narragansett) and minor non-clastic sediments. Metamorphism is absent or very low but occasionally reaches higher grade locally near intrusions (e.g. southwestern Narragansett Basin). Generally undeformed to minor deformation.

Uranium endowment - generally low to moderate but may locally be high (conglomerates, coarse sandstone, coal, etc.). Local concentrations possible.



2-BINARY OR TWO-MICA GRANITES

Various ages - generally Ordovician to Permian

Composed of intrusive igneous rocks of granitic composition with significant (> 1%) amounts of muscovite mica. May contain small amounts of intrusive rocks of other types and compositions and xenoliths of country rock. Metamorphism absent to low grade granite rocks of medium to high grade are listed under granitic gneisses (§). Deformation is variable, many show well developed foliation, others are undeformed. Most are thin sheet-like bodies (< 1 km. in thickness) occurring in areas of medium to high grade metamorphic rock.
Uranium endowment - moderate to very high, local uranium concentrations.



3-GRANITES OF CALC-ALKALINE COMPOSITION

Various ages - Precambrian to Cretaceous
Composed of intrusive igneous rocks of granitic composition consisting of quartz, potassium feldspar, plagioclase, biotite and/or hornblende. Granites with significant muscovite are not included (see Binary Granites 2). Granites with large amounts of alkali feldspar, sodic amphibole or other indicators of high alkali content are listed under Alkalic Plutonic Rocks 4.

Metamorphism is absent to low. Deformation is variable. Bodies of granite range from small sheet-like units to large batholith size units of unknown thickness.
Uranium endowment - moderate to high, dispersed, usually no major local concentrations.



Figure 3-3. EPA Generalized Bedrock Map of New England



4-ALKALIC PLUTONIC ROCKS

Various ages-Precambrian to Cretaceous

Composed of intrusive igneous rocks rich in sodium and potassium indicated by large amounts of alkali feldspar or other alkali rich phases (e.g. riebeckite). Includes alkali feldspar granites, riebeckite granites, alkali feldspar quartz syenites, alkali feldspar syenites, quartz syenites, and syenites. Metamorphism absent to low grade. Deformation absent to moderate. Generally small to moderate size batholiths and stocks. Uranium endowment—moderate to very high, generally dispersed, occasional local concentrations.



5-PLUTONIC ROCKS OF INTERMEDIATE COMPOSITION

Various ages-Precambrian to Devonian

Plutonic igneous rocks of intermediate composition, including granodiorites, tonalites, monzonites and quartz monzonites. Metamorphism absent to low grade (medium and high grade intermediate rocks are shown under intermediate and mafic gneisses 6-9). Deformation variable. May contain plutonic rocks of other compositions. Bodies are small to large stocks and sheet-like units. Sometimes associated with granitic gneisses. Uranium endowment—low to moderate, dispersed.



6-MAFIC PLUTONIC ROCKS

Various ages-Precambrian to Cretaceous

Plutonic igneous rocks of mafic composition including gabbros, diorites, monzonodiorites, and quartz diorites. May contain small amounts of rocks of other compositions. Metamorphism absent or low grade. Deformation variable but generally low. Bodies are small to moderate size stocks and sheet-like units. Usually associated with other plutonic rocks of intermediate to granitic composition. Uranium endowment—very low to low, dispersed.



7-GNEISSES—GENERAL

Various ages—dominantly Precambrian and Ordovician

Rocks with moderate to well-developed foliation and gneissic texture. Compositions highly variable but generally derived from sedimentary rocks, but also includes some igneous rocks. This does not include stratified gneissic units except in a few instances where mappable at this scale. Also does not include gneisses of probable plutonic origin. Often associated with migmatites and plutonic rocks of intermediate to granitic composition. Bodies are small to large units usually fault bounded or showing intrusive contacts. Generally in elongate bodies conforming to regional trends. Metamorphism is medium to high grade. Deformation is moderate to intense. May represent uplifted or remobilized basement rocks. Uranium endowment—variable, low to high, depends on rock composition and metamorphic processes. Locally may be very concentrated but not mappable at this scale.



8-GNEISSES OF GRANITIC COMPOSITION

Various ages—generally Precambrian and Ordovician

Gneisses of granitic composition and probable plutonic origin. Moderate to well developed foliation and gneissic texture. May include granite, granodiorites, migmatites and gneisses of other compositions. A few stratified units are shown but most are intrusive bodies or fault bounded. In general, they are slightly elongate and follow regional trends. Metamorphism is medium to high grade. Deformation is moderate to intense. Probably represent deformed and metamorphosed intrusive bodies. Uranium endowment—moderate to very high, generally dispersed but local concentrations possible.



9-GNEISSES OF INTERMEDIATE TO MAFIC COMPOSITION

Various ages—Precambrian to Devonian

Gneisses of intermediate to mafic composition, generally of plutonic origin. May contain plutonic and gneissic rocks of other compositions. Small to moderate size bodies, most are elongate and conform to regional trends. Metamorphism is medium to high grade. Deformation is moderate to intense. Uranium endowment—very low to moderate, generally dispersed.



10-ULTRAMAFIC ROCKS

Age—Cambrrian to Silurian

Rocks of ultramafic composition including serpentinites, norites, harzburgites, troctolites, etc. Generally small bodies moderately to strongly deformed. Metamorphism is variable. Some may represent fragments of oceanic crust. Uranium endowment—very low



11-STRATIFIED METAMORPHIC ROCKS

Age—Precambrian to Devonian

Metamorphic rocks consisting of sequences of marine and non-marine clastic and non-clastic sedimentary rocks, volcanic rocks of mafic to felsic composition and other rock types. In general, variations in lithology are too small to be mappable at this scale. Metamorphism and deformation are highly variable, but generally are lowest in northern Maine, and increase to high metamorphic grade and intense deformation in southwestern New England; although other areas of high grade metamorphism are found throughout New England. Bodies of plutonic rocks and gneisses too small to be mapped at this scale are also included. Uranium endowment is highly variable and is dependent on rock type, metamorphic grade and the types of plutons in the vicinity. Local high uranium contents can occur. These variations can not, in general, be mapped at this scale.

Figure 3-3 (continued)

Local variations in soil type over the same rock can lead to large differences in indoor radon levels.

A study by the Regional Air Pollution Control Agency of Dayton, Ohio found the uranium content of Ohio soils as low to average but some are highly permeable to gas. Homes built on gas permeable substrates such as sand, gravel or fractured bedrock had higher readings (60).

Pathways

Pathways of radon into structures are determined in part by construction type. Features which allow free passage of radon gas contribute to high indoor levels. A number of structural components affect indoor radon levels including basements, crawlspaces, sump holes, foundation types, tightness, and number of levels.

Higher radon readings have been associated with houses containing basements, crawl spaces, sump holes, and above average amounts of insulation in addition to certain soil factors. In the above Ohio study the average concentration in houses with basements was 8.4 pCi/l compared with 4.1 pCi/l in houses with slabs (EPA's sampling protocol involves testing which includes the lowest potentially habitable area of a house). Houses with crawl spaces averaged 10.3 pCi/l compared to 3.5 pCi/l in those without crawl spaces. This is possibly because there is seldom any barrier between the crawl space and the floor. Other studies have found houses with vented crawl spaces to have lower radon concentrations than those with slab-on-grade construction or basements. Houses with sump holes averaged 12.2 pCi/l compared to 4.7 pCi/l in those without. Sump holes go through the slab and are in direct contact with the substrate beneath (53, 60).

In a Connecticut study foundation type predicted radon levels, with higher readings in homes with cinder block foundations as proposed to poured concrete and possibly cellars with dirt floors (60). Cinder blocks are more porous than concrete. Radon concentrations have been found to be lower; in multilevel than single story houses; in non-airconditioned houses (more ventilation); and in houses built without masonry (a radon source) (53).

One earth-encased concrete house in Beverly, Massachusetts was tested at the request of DOE which considered it an outstanding example of energy conservation techniques. Radon concentration was measured at 200 pCi/l, 50 times the EPA action level. Of the 4 components assessed as contributing to high radon levels- uranium content of the soil, porosity of the soil, pathways into the house, and ventilation - uranium content and soil porosity were not unusually high. When a ventilation system and heat exchanger were installed radon concentration dropped to 2 pCi/l (61).

Ventilation and Pressure Differential

Ventilation rates are another factor affecting radon levels in structures. A simple test performed during a meeting of this Commission indicated a reading of 1.6 pCi/l during the first hour when the ventilation system was left off. Shortly after the system was turned on readings began to drop. By the end of

the second hour the room contained only 0.6 pCi/l. The movement of outdoor air through a structure will dilute and remove radon gas thus lowering its concentration.

However, negative pressures created by mechanical or passive ventilation or indoor/outdoor air temperature differentials can create negative pressures which promote the entry of radon bearing soil gas into the structure.

Habits and Activities of Occupants

Activities of occupants which can increase indoor radon levels include heavy use of showers or washing machines, or performing other tasks in which water is agitated. Concentrations of airborne radon are also affected by patterns of opening windows and the presence and frequency of use of whole house, bathroom or kitchen fans, stove hoods, wood stoves, oil or gas burners, or clothes dryers.

RADON PATHWAYS INTO STRUCTURES

Radon may enter structures as a gas from uranium-containing rock or soil, dissolved in well water emerging from taps and showers, or to a lesser extent, in building products made from such rock.

Airborne Radon

It is a principle of physics that a gas will move from an area of high concentration or pressure to one of lower pressure. Thus radon in the cracks of rocks in an underground mine will move into the air of the mine cavity and, if a passageway exists, radon in the soil or rocks beneath a structure will move into the structure.

Radon gas may enter a building through cracks or gaps in a basement or foundation, through dirt floors and holes from form ties and sumps Fig. 3-4 (49). Soil is the major source of radon in indoor air. This is supported by the fact that the parts of buildings closest to the ground have the highest levels. For example, in 85 homes tested in a University of Maine study basements had twice the radon concentration of other living areas and 8 times the concentration of ambient air (50).

Radon in Water

Surface water concentrations of radon are low and are further reduced by exposure to air since radon simply moves into the air. However, well water which has passed through uranium bearing rock can carry radon into a building. This radon can be ingested when people drink or prepare food with such water,

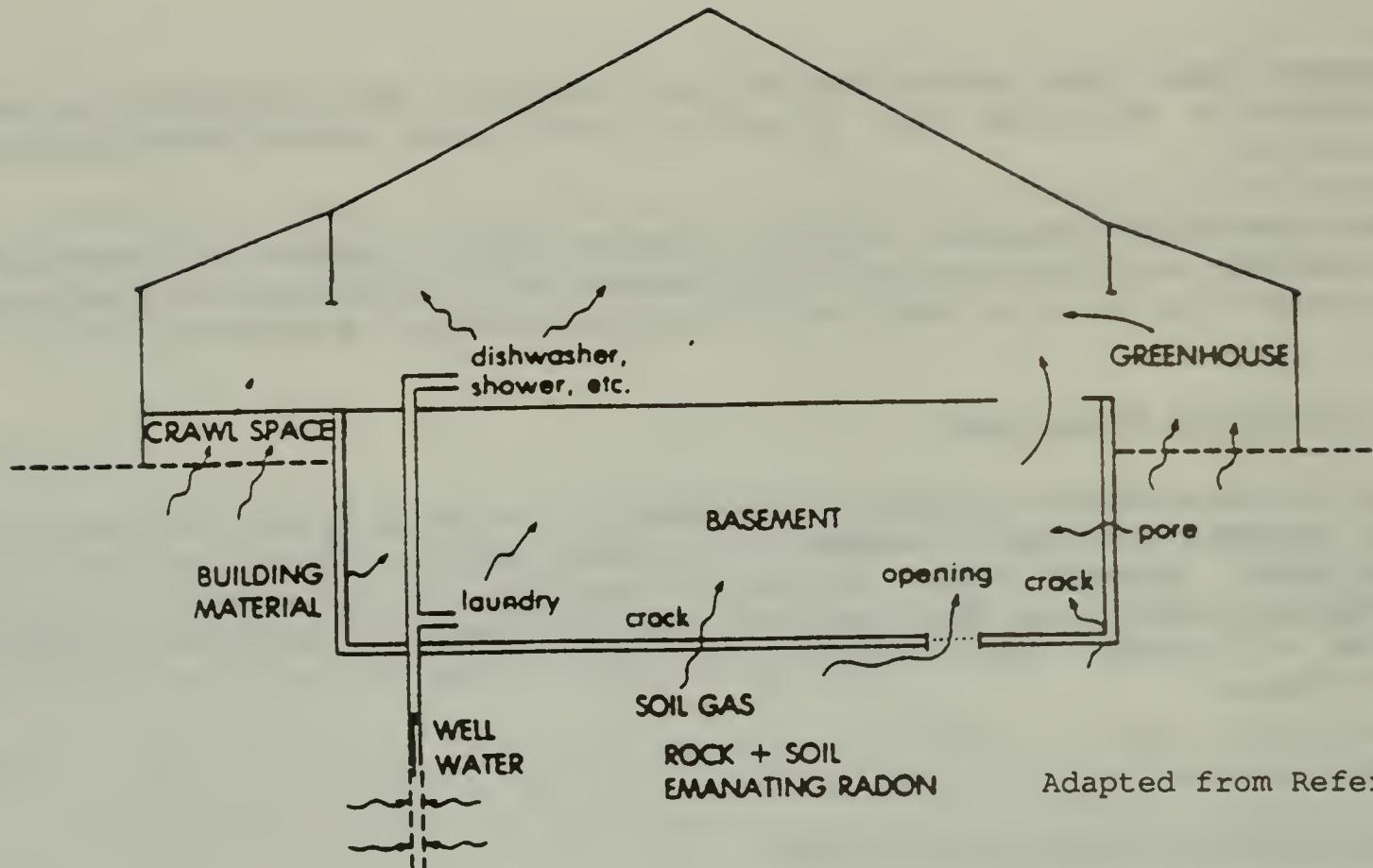


Figure 3-4. Pathways of Radon into Structures (50)

however, inhalation of radon and its entry into the lungs is of greater concern. As water emerges from taps and showers radon is released into the air of a kitchen, bathroom or laundry. Agitation increases the rate at which radon is emitted.

Other Sources

Radon is emitted by concrete or brick made from uranium bearing rock or sand (8,9). Building materials are not considered a major source of radon (49) but may contribute significant amounts in certain cases. Examples are: solar heated structures with rock used for heat storage; earthen homes; and large stone fireplaces or walls. More soil and rock is used in their construction (62). Small amounts of radon can also enter structures in coal, oil or natural gas and be emitted from poorly vented cooking or heating systems.

RADON IN STRUCTURES: SCOPE OF THE PROBLEM

Indoor radon has been reported from Finland (51) and Canada. In the United States elevated levels have been found in every state. While the average readings in a state or region were often low, in many cases a significant fraction of the buildings had readings above the EPA action level of an annual

average reading of 4 pCi/l. Since the country has not yet been thoroughly surveyed using statistically valid techniques it is unknown how many areas with high levels exist. Of almost 10,000 homes in Canada, 264 (3%) were above 4 pCi/l and 49 (0.4%) were above 10 pCi/l (25).

Federal and Nationwide Studies

A study by Lawrence Berkeley Laboratories for the DOA included a compilation of field data. Analysis from 20 studies covering a total of 1377 homes from 38 regions of the country where there was no previous expectation of high radon levels indicated an average reading of 1.5 pCi/l, and 1-3% of the homes had levels of 8 pCi/l or more. Homes with the highest levels were located in eastern Pennsylvania, Fargo, North Dakota, the Spokane River valley of eastern Washington and northern Idaho; and parts of Colorado, Idaho, Montana, Tennessee, Maryland, Florida and Maine. Few used statistically based sampling procedures but most selection processes had a strong random component, e.g., among employees of a particular institution, or participants in an energy conservation program, although about 1/3 were done because of prior information suggesting a probability of high radon concentrations (55, 63).

Terradex Corporation of California measured radon levels in the usual living areas of homes which are not associated with man-made sources of radium or uranium contamination. Alpha track detectors were used. Over 30,000 homes were tested. The data is grouped by 5 regions labeled northeast, midwest, northwest, southeast and mountain states. Measurements were made from 1980 through December 1986, and each varied from a week to a year. In some cases more than 1 detector was placed in a house and time of year for the measurements varied (25).

Data was accumulated from Terradex clients and is, therefore, not random. Larger numbers of readings are from known hot spots in New Jersey, Maine and Pennsylvania. Most of the very high measurements came from the northeast region which includes the above 3 states, along with Massachusetts, Vermont, New Hampshire, Connecticut, Rhode Island, New York, Delaware, Maryland, and the District of Columbia (25).

The northeast has the highest average, 11.1 pCi/l, considerably higher than the mountain states with 6.5 pCi/l and more than twice the midwest average of 5.1 pCi/l. Notably, both the northeast and mountain states contain granitic mountain ranges. Forty-four percent of the homes in the northeast had readings greater than the 4 pCi/l EPA action level, and 25% had readings greater than 8 pCi/l. Pennsylvania had the highest percentage of homes with readings above 4 pCi/l (56%) (25).

When the New England states are considered separately, Rhode Island has the highest average reading at 12.4 pCi/l and the greatest number of homes with readings above 4 pCi/l (30%). The Rhode Island average, however, is based on relatively few readings - only 20. Massachusetts has the lowest average at 2.4 pCi/l, with 17% of homes above 4 pCi/l (25).

EPA initiated systematic collection of data on radon levels in single family residences during the winter of 1986-87. A cooperative program was developed between EPA and 10 states (64). Over 20% of the 11,600 homes tested contained

radon levels above EPA's action level. The percentage of homes above 4 pCi/l was highest in Colorado (39%). The other states and their associated percentages were: Wisconsin (27%); Wyoming (26%); Kansas (21%); Rhode Island (19%); Connecticut (19%); Kentucky (17%); Tennessee (16%); Michigan (9%); and Alabama (6%). It is notable that the 2 states with the lowest percentages of homes with readings over 4 pCi/l had the 2 highest single readings - Alabama with 180 pCi/l and Michigan with 162 pCi/l. This suggests that areas with low average amounts of radon might still have hot spots (5,64). The data suggests all 10 states may have a significant fraction of homes with radon levels of concern (64). The worst hot spot identified is Clinton, New Jersey, a town of 2000, where 105 homes sited above a uranium-laden limestone cliff had readings over 4 pCi/l. Thirty-five of these measured over 200 pCi/l and 5 were above 1000 pCi/l.

Statewide and Regional Studies

The New Jersey Department of Environmental Protection study showed a statewide average of 5.4 pCi/l, with measurements ranging from 0.1 to 246 pCi/l. Thirty-three percent of homes had levels greater than the EPA action level implying need for further testing or remedial action. The state averages are elevated by the high readings along the Reading Prong. One hundred and sixty-five public buildings were also tested including schools, hospitals and municipal buildings around the state. Radon levels were found to be generally lower than in homes, but 13% were above 4 pCi/l (65).

In the Ohio Regional Air Pollution Control Agency study 163 locations were sampled beginning in early 1986. The average radon concentration was 7.0 pCi/l, with 21% of homes above 10 pCi/l (60).

The BPA monitored radon levels in over 250 homes in Washington, Oregon, Idaho, and Montana for several months. First floor living areas averaged 1.20 pCi/l with levels ranging from 0.05 to 15.0 pCi/l. Four percent of these had levels above 5 pCi/l (5).

New England Studies

Of 85 homes tested by the University of Maine 30% exceeded EPA's action level. Elevated radon levels in water were found in some homes (50).

The joint survey of radon levels in indoor air and well water by the Connecticut Departments of Health Services and Environmental Protection assessed the predictive value of hydrologic and household parameters, and the relationship of radon levels in the well water to levels in indoor air. EPA's method of water sampling was used in 262 homes and alpha track devices were placed in the living areas of 202 of these.

Radon levels in indoor air ranged from 0.1 to 24.6 pCi/l, with a mean of 2.0 pCi/l. 11% had levels of 4.0 pCi/l or greater. Well water ranged from 100 to 130,240 pCi/l. Twenty-six percent of wells had levels of 10,000 pCi/l or above. Regression analysis indicated 17% of airborne radon was attributable to water.

RADON IN MASSACHUSETTS

Uranium Content of Rock and Soil

As indicated previously EPA studies of the bedrock geology of Massachusetts suggest a potential for high indoor radon levels in some areas. Uranium content of rocks and soils may vary in different parts of the Commonwealth due to different bedrock types, e.g., granites tend to be higher in uranium content than the sedimentary rocks found in Boston. However even rock types with generally low uranium levels may have localized pockets or "hot spots" (59).

Indoor Radon

Several thousand buildings, mostly homes, have been tested for radon in Massachusetts though again most of the buildings were not randomly selected. Massachusetts homes tested in the Terradex study averaged 2.4 pCi/l, however 17% were above the EPA action level.

The University of Pittsburgh measured radon in homes in Middlesex and Essex counties in Massachusetts. The average readings in the main living areas were similar to the 1.4-1.5 pCi/l found in other studies, and were lower than around the Reading Prong and some southern and western states. However, 15% of the houses had readings of 3 pCi/l and over, and 1% were 15 pCi/l or above (61). Some very high readings were found including the earth-encased concrete house in Beverly.

Boston's Channel 7 (WNEV), a CBS affiliate, sponsored a project in which homeowners throughout Massachusetts were invited to participate in a radon testing program. Fifty-three (30%) of the 174 readings were above 4 pCi/l; 9 readings (5%) were above 20 pCi/l (66).

The DPH Radiation Control Program (RCP) has a database of approximately 3000 sample results. Again 14 to 25% of the readings are above EPA's action level of 4 pCi/l and 1% are above 20 pCi/l. Thus far no hot spots have appeared (67).

While none of the above may have been random samples it is notable that 10-30% of the readings were above the action level (assuming the University of Pittsburgh data to be 10%). Taking 20% for purposes of argument this could suggest 320,000 homes in the state might have elevated radon levels. Since the samples were non-scientific and only 3000-4000 readings are on record a large degree of uncertainty remains about the scope of elevated indoor radon in Massachusetts.

Radon in Water

There appears to be little problem with radon in public water supplies in

Massachusetts. In 1981-1982 EPA and DPH studied groundwater fed public water supplies in 100 towns covering 90% of Massachusetts residents who drink public groundwater. The water was virtually free of radon. Many towns using groundwater store it in standpipes or reservoirs where the radon dissipates (61). The Quabbin Reservoir has never shown a radon problem (68).

RISK ASSESSMENT FOR RADON EXPOSURE

Early findings of elevated indoor radon levels prompted questions regarding associated health risks. Higher rates of lung cancer occur in uranium miners with long term exposure to high levels of radon than in the general population. Risk estimates are based on extrapolation from high dose exposures, particularly of miners (50), to low doses.

Hazard Identification

While evidence clearly shows lung cancer is greater in miners than non-miners, there is disagreement whether this is due to radon alone or is enhanced by other factors. Other radioactive elements such as uranium are present in mines, and the air contains dust particles which may lead to chronic irritation and predispose miners to lung cancer (68).

The situation in mines also favors attachment of radon progeny to particles (68). In addition miners tend to be male, and also to be smokers. Possibly the combination of radon, smoking and dust produced the higher cancer rate (5). It also has been argued that lung damage occurs over an extended period and often isn't distinguishable from the ravages of other ailments (26).

Radon clearly produces radioactive particles which damage bronchial tissue in the lungs (26). Evidence for the hazardous nature of radon also comes from its short half-life of 3.8 days and the fact that during decay it emits the same alpha, beta and gamma radiation as other radioactive elements. The effects of radiation exposure are well known (55) from Japanese atomic bomb survivors, and industrial workers (50).

Dose Response

Long term effects of low doses of radiation are still largely unknown. However, a DPH study found elevated cancer levels in 5 towns around the Pilgrim Nuclear Power Plant in Plymouth where residents are subjected to continuous low level radiation. This has stimulated the National Institutes of Health (NIH) to study people in the vicinities of all nuclear power plants in the United States. Early experiments with low radon levels did not lead to lung cancer because only radon was used and not radon progeny. However, recent human cell and animal experiments suggest low radon levels cause cancer (50). There is as yet no direct evidence of a link between low level radon exposure and lung cancer in humans (26).

Experiments to determine the biological effects of radon inhalation usually involve acute lethal and sub-lethal exposures so that effects will appear

within reasonably short periods of time. Generally sublethal doses shorten life and cause stunted growth - similar to the effects seen from exposure to other types of radiation (68). Risk assessment for radon exposure is based on: knowledge of how radiation affects tissue; known effects of high radon doses; assumptions about extrapolation from high to low doses (50).

Risk estimation assumes that risk of lung cancer is proportional to both dosage and total exposure. All radon risk estimates are uncertain due to dose-response models which are uncertain for low levels (9). The unknown factor is whether the risk from low doses is purely proportional to the risk at high doses (linear), e.g., does someone with 1/10 the dose have 1/10 the risk? Or, is there a threshold - a level below which there is no risk (50). An NAS committee on the topic feels the linear model is correct for both air and water borne radon.

Risk estimates below assume the linear model. There is such uncertainty however that risks may be half or double the estimates (50). To account for this estimates are often given as a range of numbers (49).

Exposure Assessment

The levels miners were exposed to were high, nearly 100 times those in the average home (5). Studies of miners with total exposures comparable to, e.g., 60 years in a house at 10 pCi/l or some other combination of dose and time, clearly show that lung cancer occurs. The lowest rate at which there is direct evidence of cancer is at about 1/10 this total exposure, which would be equivalent to 20 years at 2 pCi/l (50). People in homes with 20 or more pCi/l have the same WLM exposure as miners before controls (55).

There is widespread occurrence of indoor radon levels above 4 pCi/l with some as high as several thousand pCi/l. Some people are exposed to the lower range of the doses miners experienced. Many more are exposed to low radon levels for years or decades. It is likely that hazardous dosages and exposures are occurring among large numbers of Americans.

Risk Characterization

Estimates of the risk of dying from lung cancer as a result of radon exposure are based on models which assume a dose over a period of time and take into account the fact that people only spend a portion of their time in the structure in question. For example, an EPA model assumes 75% of the time is spent at home for a lifetime of 70 years. As Table 3-2 shows at 4-20 pCi/l 1-5 out of every 100 people would be expected to die from lung cancer. At 20-200 pCi/l 6-21 deaths would occur and at 200 pCi/l 44-77 deaths. In the latter case if the same 100 persons lived only 10 years in a house with 200 pCi/l only 14-42 deaths would occur (49). All of these are very high risk factors. Governmental action is usually considered appropriate when the risk is 1 in 100,000. It should be noted that the EPA action level is somewhat arbitrary. Public health concern should not stop at 4 pCi/l.

About 4 out of every 100 people die of lung cancer. The Surgeon General estimates about 85% of lung cancer deaths are due to smoking (49) and EPA's

Table 3-2. Radon Risk Assessment

pCi/l	WL	Estimated number of lung cancer deaths due to radon exposure (out of 100.)	Comparable exposure levels	Comparable risk
200	1	44 — 77	1000 times average outdoor level	► More than 60 times non-smoker risk
100	0.5	27 — 63	100 times average indoor level	► 4 pack-a-day smoker
40	0.2	12 — 38		► 20,000 chest x-rays per year
20	0.1	6 — 21	100 times average outdoor level	► 2 pack-a-day smoker
10	0.05	3 — 12	10 times average indoor level	► 1 pack-a-day smoker
4	0.02	1 — 5		► 5 times non-smoker risk
2	0.01	.7 — 3	10 times average outdoor level	► 200 chest x-rays per year
1	0.005	.3 — 1	Average indoor level	► Non-smoker risk of dying from lung cancer
0.2	0.001	.1 — .3	Average outdoor level	► 20 chest x-rays per year

Source: U.S. Environmental Protection Agency

Science Advisory Board estimates that about 10% are due to radon (49). Radon could cause roughly 10,000 deaths per year. Radon in water may be responsible for about 1000 deaths each year (9). The risk of a smoker dying of lung cancer is increased considerably if the smoker is also exposed to elevated levels of radon (18).

It is useful to compare the risk of lung cancer death from radon exposure to that of smoking (Table 3-2). For example persons exposed to 4 pCi/l have 5 times the risk of a non-smoker; 200 pCi/l is 60 times the non-smoker risk and equivalent to that of a 4 pack a day smoker (49).

The above estimates could be low. An NRC study released early in 1988 (18) suggests the lung cancer risk from lifelong exposure to radon is several times greater than earlier estimates.

RADON MITIGATION

The radon problem appears to be resolvable. Unacceptably high levels of indoor radon can generally be lowered in existing structures and prevented in new ones. Homes in Pennsylvania and New Jersey reading 3000-4000 pCi/l have been improved to an annual average below the EPA action level of 4 pCi/l (5). The goal of mitigation is to reduce indoor radon to or below 4 pCi/l. Mitigation and prevention techniques, however, may vary in complexity, cost and effectiveness. In addition mitigation may be required on an ongoing basis

since structures age, settle and new cracks appear.

Indoor airborne radon levels are determined by the difference between radon's rate of entry and its rate of removal. The rates of infiltration of soil gas and escape of radon from tap water can be reduced by source control methods which prevent, deflect or reduce radon's entry. An increased removal rate is achieved through increased ventilation (5).

Source Control

As indicated earlier air which is naturally or mechanically driven from a building in an effort to lower radon concentrations lowers the inside air pressure. Pressure is also lowered by clothes dryers, the chimney effect of warm air rising through a building and out openings at the top, wind blowing around the house, and fireplaces and furnaces. When air pressure is lowered radon, which is at concentrations in the soil thousands of times greater than in the air, will be drawn in (70).

A number of treatments have been proposed or used to lower the rate at which radon enters a living space. There is little scientific information available regarding the effectiveness of some of these techniques although theoretically they would seem to be effective (50) and may require special procedures.

Venting crawl spaces should dilute radon before it enters living areas (5). Sealing untrapped drains or floors over crawl spaces, caulking cracks or openings in slab floors, and placing airtight barriers between floors and crawl spaces are straightforward techniques. A floor drain or sump pit can be vented outdoors using a dryer hose and small electric fan (51).

A system used in some houses with full basements is to sink pipes through the basement floor and connect them to an exhaust fan. This both draws radon laden air outside and offsets the house's tendency to draw air from the basement to rooms above by decreasing the pressure beneath the basement (51). Using this technique, EPA has turned the homes on the New Jersey cliff with the 10 highest readings into a research lab for mitigation methods. Of these 6 are now reading below 4 pCi/l (71).

Most public water supplies come from reservoirs where radon escapes into the air, whereas private supplies usually come from groundwater and are more likely to contain radon. Activated carbon filters, depending on type, can remove up to 99% of waterborne radon and also remove potentially toxic uranium salts. The unit is functionally similar to a water softener and contains a backflushing system to remove accumulated material from the filter. However recent research has suggested that backflushing can release radon progeny and there is concern developing about disposal of the unit.

Aeration systems are available which spray water inside a large container. Air is blown through it and the radon is vented outdoors. This can remove up to 99% of the radon but is costly.

Removal

Radon concentrations vary with the number of air changes per hour which are lower in winter when structures are kept closed. The average annual ventilation rate in single family houses has been reported at 0.5-1.5 ach (53). New homes vary from 0.2 to 0.5 ach due to their tighter construction (9). Energy efficient buildings may be as low as 0.1 to 0.2 ach (69).

Doubling the ventilation rate in relation to a fixed source rate decreases radon concentration by 50% according to the dilution principle (Fig. 2-3). Ventilation methods used to reduce radon are the same as those used in the reduction of indoor air pollution in general: passive ventilation and active ventilation in the form of fans (both exhaust and intake) and air-to-air heat exchangers (50).

Tightly-sealed houses in terms of air leakage may have high radon levels in spite of a relatively low rate of soil gas infiltration. The ventilation rate can be increased significantly if several windows are kept open a little in winter. Using ventilating fans or opening windows in bathrooms or other water use areas is also effective when water is a significant source (50). Tightly sealed houses with balanced ventilation are optimal.

Removal of airborne particles, e.g., via a fan connected to a filter, decreases the total number of particles in a room. This leaves a larger proportion of radon decay products unattached. However, there is a potential for increased lung doses of radiation from these increased numbers of unattached progeny. This might occur with any techniques employing selective removal of aerosols through filtration (53). So the net effect of filtration is unclear.

One quarter of the buildings constructed on phosphate tailings or mineral land and one-half of those constructed on uranium tailings require special construction methods (9). Controls such as special construction or alterations have a cost. Additional natural or mechanical ventilation uses more energy. Fans, heat exchangers, etc. involve capital and maintenance costs. High efficiency air cleaners for recirculated air reduce levels of particulates and associated radioactivity but are costly. Surface coatings for basement walls and floors which are being tested could be considerably less costly than other measures. A Colorado Department of Health study of effectiveness of control measures found dilution by ventilation to be most cost effective (53). Adding a vented crawl space has been suggested as the most cost effective way to minimize radon infiltration in new construction (53).

RADON: FEDERAL INITIATIVES

EPA is regarded as the lead agency for indoor air quality. Statutes under which it might regulate radon include: TSCA (aimed at toxic air pollutants); UMTRCA (residential projects on tailings); SDWA (for water-borne radon); CERCLA (for radioactive substances on hazardous waste sites); and very importantly, SARA (which in 1986 gave EPA explicit authority to conduct research and disseminate information on radon and other indoor air pollutants).

Nationwide Database

Both EPA's uranium bearing rock and soil mapping program and DOE's bismuth monitoring program are aiding predictions of where geological factors might lead to high indoor radon levels.

EPA's joint study with the states continued during the winter of 1987-88. Homes were tested in 7 more states including Massachusetts. In addition to the 10 states involved in the 1986-87 EPA study, 11 are surveying for radon on their own - Alaska, Florida, Idaho, Ohio, Illinois, Indiana, New Jersey, New Mexico, New York, North Carolina, and Virginia. The Indian Health Service is testing reservations in Michigan, Minnesota and Wisconsin (26). According to deputy EPA Administrator A. James Barnes these results and those of epidemiological studies will combine to give EPA "a robust database in another couple of years" (64).

A separate and somewhat duplicative national survey, to be conducted by EPA, was mandated by the 1986 amendments to the Superfund Law. Its purpose is to determine the distribution of indoor radon levels throughout the U.S. in homes, schools and workplaces. Detectors were projected to be in place in 2000-5000 homes across the country by late 1987 (64).

Health Effects and Risk Assessment

In terms of number of potential cancer deaths a year radon is EPA's greatest environmental problem--more severe than hazardous waste and toxic chemicals (5). EPA estimates 4 to 8 million homes in United States exceed the action level (64).

A number of epidemiological studies are underway to investigate the link between radon in homes and lung cancer. The National Cancer Institute (NCI) is undertaking projects in New Jersey and Missouri; and Argonne National Laboratory has a study in progress in Pennsylvania (64). The multi-year Argonne study is using data from homes in Eastern Pennsylvania, where particularly high indoor radon levels occur, to look at lung cancer in women (26).

EPA is developing an Integrated Risk Information System (IRIS) which is a computer-based chemical-specific risk assessment and management program. As of 1988 over 350 chemicals should be accessible. The National Air Toxics Information Clearinghouse has a complementary data base. IRIS presumes some knowledge of health sciences but not technical expertise (72).

The Office of Health and Environmental Research (OHER) has sponsored radon research for several decades including animal studies, and radon transport and diffusion into and within structures. It has also supplemented previous Public Health Service uranium miner studies with research on New Mexico miners which includes more accurate exposure data (1).

Other Efforts

EPA's Radon/Radon Progeny Measurement Proficiency (RMP) Program assists states and the public in selecting companies with competence in measuring radon

levels. Detector operations and data management quality are evaluated semi-annually. The program's objective is to promote standard measurement and quality assurance procedures (73). RMP approves laboratories as well as businesses (49).

One hundred radon-proof homes are being built in varying geological situations along the Reading Prong New Jersey. This is a joint effort of EPA, The National Association of Homebuilders Research Foundation, the New Jersey Builders Association and the State Department of Community Affairs which regulates the housing industry (44).

The National Conference of State Legislatures has received an EPA grant to provide information and assistance to states developing radon programs. The project is entitled State Radon Programs: The Role of Legislation. The grant will supply funding for research and a report on the nature and scope of the problem, outlining existing federal and state programs and presenting policy options (74). EPA's regional offices (including Boston) offer radon information and assistance and an information hotline is envisioned (9).

The Federal Emergency Management Agency (FEMA) may allow disasters attributable to radon to qualify for Federal aid under the Disaster Relief Act.

The CIAQ radon working group co-chaired by EPA and DOE, will coordinate DOE and other programs. DOE program as a whole (and EPA) is coordinated with other countries through the Commission of the European Communities (1). CIAQ and EPA have co-sponsored international conferences on Indoor Air Quality in Berlin, Stockholm and Amherst.

Standards, Regulations and Guidelines

EPA has responsibility for standards regarding air-borne ionizing radiation and has developed guidelines for homes built on uranium mine tailings (40CFR192). These guidelines suggest a maximum radon level of 4 pCi/l (or 0.02 WL of radon decay products) (9).

There are currently no federal regulations or guidelines for indoor radon levels for structures built on natural soil (9). EPA recommends trying to reduce radon levels to annual average exposure of 4 pCi/l (0.02 WL) or lower (49) and taking action if levels are above 4 pCi/l or exposure approaches 1 WLM/year (51) and suggests that the higher the radon level, the greater the urgency to confirm the reading and take action (Table 3-1) (9).

EPA also proposes that certain factors be considered in determining tolerable indoor levels, e.g., the presence of smokers or children, the percent of time spent at home, whether someone sleeps in the basement, how long the family expects to live in the home. Tobacco smoke contains particulates to which radon progeny may attach, and children have shown greater sensitivity to other radiation types. EPA recommendations include: discouraging smoking, spending more time in areas with lower radon concentrations, opening windows and using fans, keeping crawlspace vents open as well as long term solutions (49).

Exposure guidelines have been criticized as arbitrary and misleading because there is evidence that level of radon progeny in the air can double if any of

the inhabitants smoke. In the absence of particulates ions adhere to walls and furniture where they pose no health threat. But if ions adhere to smoke particles they are slowed down by a factor of 10 and may be inhaled (75). The EPA recommended maximum level was set in part because EPA feels it is difficult to reduce levels much below 4 pCi/l (64).

A range of guidelines and standards has been proposed for indoor radon by other (5). Unlike statutes and regulations, guidelines do not carry the force of law. Standards may either be advisory or carry regulatory force (if referenced in legislation). In 1984 the National Council on Radiation Protection and Measurements (NCRP) recommended 0.055 WL in existing buildings and suggested remedial action at greater than 2 WLM per year (8 pCi/l) (76). The World Health Organization has established 0.11 WL for existing structures (9). ASHRAE recommends 0.01 WL in new buildings. ASHRAE has also proposed ventilation standards which have been adopted as a minimum in Sweden, Denmark and France. No standards have been set for water-borne radon (55).

RADON: STATE, MASSACHUSETTS AND LOCAL INITIATIVES

A number of states have taken steps to better define the extent of elevated radon levels. In most cases action has been the result of legislation requiring a statewide survey. Alaska, Idaho, New Jersey, New Mexico, New York, North Carolina, Florida, Illinois, Ohio, Indiana, and Virginia are currently "surveying" (26,64).

A few states have gone further. New Jersey began a \$4.2 million comprehensive initiative in 1986. New York and Pennsylvania are spending similar amounts. New Jersey, which estimates 1.9 million homes may have unsafe levels, has the most comprehensive state program to date, calling for a state radon survey and an epidemiological survey to determine if radon causes cancer. Homes of 1200 living cancer patients will be tested. The program will also include confirmation of test results through free second opinions, monitoring, certification of home improvement and testing firms, and an information and outreach program (26).

The number of firms in New Jersey offering radon testing increased from 3 in 1985 to 120 in 1987. There were none doing mitigation in 1985, 22 in 1987. The state oversees and regulates testing firms. New Jersey has strict licensing and certification standards. About half the applicants for certification have been rejected. The state offers free second opinions of test results. New Jersey and Pennsylvania have toll-free radon hotlines staffed by scientists. Both have produced pamphlets and brochures. New Jersey is doing periodic public opinion surveys. In northern New Jersey 80% of real estate transfers involved a radon test in mid-1987.

Pennsylvania and New Jersey are also providing low interest loans to assist home owners with modifications to reduce radon. The Pennsylvania program, which has been in existence for 2 years, offers \$3 million - up to \$7,000 per home with graduated interest rates based on income and free radon tests to homeowners on the Reading Prong (5,77).

Radon Testing

There is currently no structured radon testing program in Massachusetts and all sampling is based on consumer requests. Those contacting RCP are provided with a list of testing firms (61).

While DPH requests that homeowners report test results, such reporting is entirely voluntary. Some contractors are providing results to the RCP, however, location is identified only by zip code. RCP has built a database of around 3000 reported sample results.

Reports of elevated radon levels (> 4 pCi/l) receive a graded series of responses, adapted from EPA's recommendations (Table 3-2). Readings are confirmed with charcoal measurements. An alpha track detector is then installed for several months to a year. When results above 20 pCi/l are obtained grab samples may be taken, and water tested in homes with private wells. Above 4 pCi/l remedial action is recommended.

Table 3-3 DPH Response to Indoor Radon Levels

Radon Concentration	Radon Progeny Concentration	Response
pCi/l	WL	
$>4-20$	$>0.02-0.1$	reading confirmed with charcoal measurements; alpha track detector then installed for 1 year.
21-100	$>0.1-0.5$	site visit to take charcoal and grab sample readings; alpha track detector installed for 3 months; if private well, water tested.
>100	>0.5	as above but alpha track detector may be left in place for a shorter period; remedial action recommended.
21-200		

(49)

RCP has reprinted a radon booklet published jointly by the EPA and the Centers for Disease Control and is also distributing 20,000 booklets and fact sheets and giving presentations to local Boards of Health, the media, realtors and others (49).

EPA's Eastern Environmental Radiation Facility has been utilized to analyze air and water samples. However, as demand increases, federal facilities are becoming less available to states. The State Laboratory Institute's facilities might be utilized but these are in need of considerable additional equipment and supplies, including charcoal canisters and scintillation vials (to analyze water samples).

Joint EPA - DPH Study

DPH regards radon as a serious health problem and, in conjunction with EPA, is undertaking a survey of radon levels in Massachusetts residences. The survey's purpose is to determine the scope of the radon problem in the state and to develop appropriate response mechanisms. Through the efforts of this Commission \$90,000 was appropriated in the Commonwealth's fiscal year 1988 budget for this purpose.

EPA is providing technical guidance, charcoal canisters, analysis of samples, and training for the telephone interviews. DPH has contracted with the University of Massachusetts Center for Survey Research to conduct the study. The Center is identifying geographic regions where clusters of homes may have elevated radon levels, distributing charcoal canisters to and conducting telephone interviews with over 2000 homeowners, notifying homeowners of results, and providing a report. DPH is overseeing the project and providing technical assistance and review. The Commonwealth controls the rights to confidentiality of the data.

Massachusetts: Standards, Regulations and Guidelines

There is currently no research underway regarding health effects of radon exposure and there are no state radon regulations or water testing standards. Local BOH regulate radiation and have the authority to promulgate regulations but are awaiting guidelines. BOH may also make a determination of whether a house is habitable, i.e., if there is a public health risk. DPH has received requests from BOH for assistance in radon study design and is providing presentations and literature.

RADON: COMMISSION RECOMMENDATIONS

Evidence that Massachusetts, as other states, has a potentially large number of buildings with health threatening levels of radon led the Commission to deviate from its action plan of assessing categories of indoor air pollutants prior to making recommendations.

Parts of the state are underlain by granitic bedrock with moderate to high uranium content. In addition a significant fraction of the several thousand indoor readings which have been made show radon concentrations above EPA's action level.

Thus, in mid-1987 when Massachusetts was among 7 states under consideration to participate in EPA's radon survey program, the Commission endorsed the proposal and was instrumental in incorporating the necessary funding in the FY 1988 budget. The Commission has stressed the need for statistically reliable data which can best be obtained through sampling a large number of randomly selected buildings.

Results of this study will be used to make further recommendations. In the interim the Commission is urging DPH and DEQE to educate the public regarding radon.

Chapter 4: FORMALDEHYDE

FORMALDEHYDE AND ITS SOURCES

The Compound Formaldehyde

Formaldehyde is the simplest member of the chemical group called aldehydes, and consists of one oxygen and two hydrogen atoms attached to a carbon (Fig. 4-1) (78). Like all aldehydes formaldehyde occurs as a vapor at room temperature. Formaldehyde is a colorless gas with a characteristic pungent odor. Although some sensitive individuals can detect the odor of formaldehyde at approximately 0.05 ppm most people only detect concentrations of 1.0 ppm or greater (79). Formaldehyde is highly reactive and dissolves in water, methanol and other substances (44).

Formaldehyde occurs naturally as an intermediate product in animal metabolism and is produced in the decay of animal and plant matter (80) and the combustion of fuels such as wood and oil.

Ambient air may contain formaldehyde from biological decay, vehicle exhaust and industrial emissions (80). Indoor air additionally may contain formaldehyde from various biological and industrial uses, numerous manufactured products, and as a result of combustion of fuel or cigarettes. Formaldehyde has been utilized as a preservative and disinfectant in hospitals and biological laboratories for 100 years (81). As a preservative it is often used as formalin, a mixture of 35-50% formaldehyde in water (44,78).

Most formaldehyde is produced inexpensively by laboratory methods. Formaldehyde is used or occurs in many consumer products either alone (free) or as part of other compounds. Half the formaldehyde produced today is utilized in the production of urea formaldehyde (UF) and phenol formaldehyde (PF) resins which are in turn used as bonding and laminating agents in wood products, in wall insulation as UFFI, and for modifying the properties of textiles or paper (Table 4-1) (44). Each of these products emits formaldehyde, at a rate dependent upon the nature and properties of the source. For example, emissions from UFFI decrease over time while emissions from cigarettes are constant.

Figure 4-1

The Structure of Formaldehyde

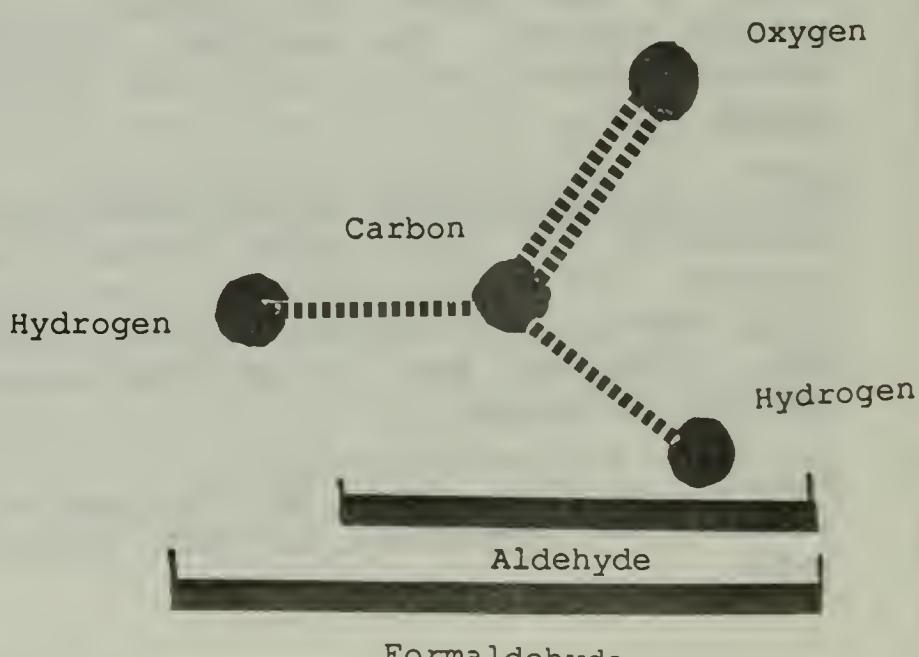


Table 4-1 Common Sources of Formaldehyde

• Acrylic	• Electrical connectors	• Plastic
• Air and furnace filters	• Electronic equipment	• Plumbing fixtures
• Air fresheners	• Embalming agents	• Plywood
• Aircraft parts	• Examining table paper rolls	• Portable tools
• Antihistamines	• Facial tissues and napkins	• Preservatives
• Antiperspirants	• Fan blades	• Pressed wood furniture
• Antistatic agents	• Faucets	• Pressure gauge bodies
• Ashtrays	• Fiberboard	• Primer coat for automobiles
• Automotive brakes	• Flour preservative	• Radiators
• Barber and beauty shop disinfectants	• Formica	• Radio and TV bases and housing
• Binders	• Glues	• Rayon
• Biocides	• Hair-setting solutions	• Roofing
• Blanket controls, bases and covers	• Hair-waving preparations	• Sewing machine parts
• Brake drums	• Hand pumps	• Shampoo
• Cigarette smoke	• Hardboard	• Soap dispensers
• Coated papers used for cartons and labels	• Hardware	• Softwood plywood
• Coatings	• Heat sensor switches	• Sporting goods
• Coatings for appliances	• Hospital bed sheets	• Stove and refrigerator hardware
• Compactors	• Insecticides	• Textile waterproofing
• Cookware handles and knobs	• Kidney dialysis procedure	• Tire rubber
• Counter and table tops	• Knobs and buttons	• Toilet seats
• Dental bibs	• Lawn and garden equipment	• Toothpaste
• Dental filling	• Maraschino cherries	• TV/radio/stereo cabinets, door panels, store displays, kitchen cabinets
• Deodorants	• Mascara and other cosmetics	• Utensil handles
• Detergents	• Nail hardener	• Vaccines
• Diaper liners	• Nail polish	• Vacuum cleaner parts
• Dinnerware	• Oil-based paints	• Water-softening chemicals
• Disinfectants	• Orthopedic casts and bandages	• Wax and butcher wet strength paper
• Door panels	• Orthopedic procedures	• Wheat grains and agriculture seeds
• Drapery and upholstery fabrics	• Paint and wood finishes	• Wool
• Drinking milk	• Particleboard	
• Dyes	• Permanent-press cotton	
	• Pesticides	
	• Pharmaceuticals	

Bonded Wood Products

Synthetic resins containing formaldehyde are used mainly as adhesives in bonded wood building materials including paneling, plywood and particleboard, wallboard, hardboard partitions, and ceiling panels (44). UF resin, a prepolymer made from a mixture of urea, formaldehyde and water, is the most common adhesive (82). Heating (curing) causes numerous different formaldehyde emitting compounds to form (83). All bonded wood products with UF resin release formaldehyde.

Release of unreacted formaldehyde from UF bonded wood products has been known since Fahrni invented particleboard in 1943. However exposure risk was at first low because the products were not widely used. Over the next several decades concern increased over the loss of forests and a dwindling wood supply. Bonded wood products found a larger market because they allowed more efficient use of harvested trees. Formaldehyde release problems arose when use of particleboard and hardwood plywood paneling increased (81).

In 1973 teachers refused to work in a new classroom building in Karlsruhe Germany because of an irritating odor. The problem was found to be large amounts of particleboard plus the fact that the ventilation system had been turned off for several weeks. Similar problems have been reported in other countries (81). Particleboard was introduced about 10 years later in North America than in Europe - since there were larger forest reserves (83).

Over the past 20 years bonded wood products have been used more and more instead of whole wood. There are three major types: particleboard, medium density fiberboard (MDF) and hardwood plywood. Particleboard is composition board with 6 to 10% resin by weight, along with small wood particles and additives. Over 90% of the resin is UF. Seventy percent of particleboard is used in cabinets, furniture, fixtures, etc. with the remainder for construction, e.g., decking or subflooring - in mobile as well as conventional homes (83).

MDF presently contains only UF resin which is 8-14% of its weight. Ninety-five percent of MDF is used for doors, furniture, cabinets and fixtures. MDF and particleboard differ in that MDF particles are separated via cooking or shredding into fibers less than 1 mm long. MDF is fine, appears more like wood than particleboard, and may be worked to achieve smooth edges (83).

Hardwood plywood is laminated with formaldehyde resin which bonds the layers of wood and veneer. It is 2.5% resin by weight. About 55% of this plywood is used for paneling, 30% for furniture and cabinets and the rest for doors and flooring. Furniture often contains particleboard on the inside and veneer on the outside. Softwood plywood also contains formaldehyde (44,83).

Urea Formaldehyde Foam Insulation

UFFI is another important source of formaldehyde emission. Problems with formaldehyde in indoor air are commonly associated with UFFI. UFFI became increasingly popular during the early 1970's and was in wide use by the end of the decade (44).

Formaldehyde, especially UFFI, has led to thousands of complaints to the CPSC (79). UFFI is produced on site by the installer. UF based resin, compressed gas (usually air), a foaming agent, and an acidic hardening agent are combined to make a foam with the consistency of shaving cream. UFFI is pumped into a wall cavity through a hose. UFFI is 75% water by weight at the time it is inserted and can take up to several months to cure and become solid - depending upon temperature, ability of the cavity to vent the water, the

chemicals used and other factors. When set it has the appearance of an open-celled foam. It usually shrinks and cracks when dry, allowing air to circulate, and may eventually crumble. UFFI has generally been used to retrofit older homes (80,84).

Other Sources

Formaldehyde resins are widely used in the textile industry for clothing, linens, draperies and upholstery fabrics. UF resin is used for color fixation since it increases the adherence of pigments to cloth. The crease resistant (permanent press) characteristics of nearly all linen and cotton come from formaldehyde as do shrink and flame resistance, and water proofing (44).

Wallpapers, especially those which are prepasted or made with fibers or layers of paper are bonded with formaldehyde resins. UF resin is utilized to impart wet strength to different grades of paper. It is found in paper products such as facial tissues, napkins, paper towels, waxed paper, and grocery bags. The Food and Drug Administration (FDA) has allowed UF treated paper to contact food since 1972 (44).

Formaldehyde is used in personal care items including cosmetics, shampoos, and disposable sanitary products. It is also found in room deodorizers, starch-based glue, fiberglass insulation, ceiling tile and floor coverings (44,83).

HEALTH EFFECTS OF FORMALDEHYDE EXPOSURE

Formaldehyde is a public health issue because exposure produces numerous physiological effects and because it is virtually impossible to escape in modern living environments. While the potential for exposure to high levels of formaldehyde (several ppm) is less in the home than in industries or occupations which produce or use formaldehyde, residential exposures are also of concern because so many more people are exposed at home and more time is spent there. Formaldehyde in building materials, furnishings, textiles and paper products increases exposure (44) as does the presence of tobacco smoke.

Public concern about formaldehyde exposure initially focused on its irritant properties, and more recently on the effects of long term exposure, particularly formaldehyde's potential as a carcinogen (44).

Physiological Action

Formaldehyde is a normal metabolite in humans and other animals. It is a vital ingredient in the synthesis of essential biochemical substances, and not toxic in small quantities (85). Formaldehyde combines with water and undergoes various reactions and is present bound to other compounds. Measureable amounts of free formaldehyde are seldom found in plasma or tissue (44).

External formaldehyde can enter the body through inhalation, ingestion, or

adsorption by the skin. Ninety-five percent of the formaldehyde inhaled through the nose is absorbed in the upper respiratory tract. At low concentrations in "clean" air most of the formaldehyde is absorbed in the front third of the nose. Formaldehyde which is inhaled into the lungs may, after a series of reactions, be oxidized to carbon dioxide and exhaled or be excreted with urine. Some is incorporated into tissues. Because it is water soluble formaldehyde can easily enter the bloodstream. Formaldehyde displays similar behavior in all mammalian species (44,79).

Range of Effects

Formaldehyde has been established as an irritant in both humans and other animals. Residential and occupational exposures have reportedly resulted in prolonged eye, nose and throat irritation, wheezing, coughing, diarrhea, nausea, vomiting, headaches, dizziness, lethargy, irritability, disturbed sleep, and olfactory fatigue. Skin contact can lead to acute inflammation, irritation, contact dermatitis and hives. Exposure can lead to bronchial asthma (406) and menstrual irregularities (Table 4-2) (44,79). High concentrations can be fatal.

It is hard to relate specific health effects to specific formaldehyde concentrations because the responses and complaints of individuals vary. Response thresholds vary by whether a person is hypersensitive, healthy or diseased. Complaints of consumers can't always be ascribed to formaldehyde.

Table 4-2 Formaldehyde Concentrations and Adverse Effects:
Occupational and Residential Studies

ppm	Effect	Type of exposure
0.0—10	Nausea, eye, nose and throat irritation, headache, vomiting, stomach cramp	Residential
0.02—4.15	Diarrhea, eye and upper-respiratory tract irritation, headache, nausea, vomiting	Residential
0.09—5.6	Burning of eye and nose, sneezing, coughing, and headaches; 3 of 7 suffered from asthma or sinus problems	Occupational
0.3—2.7 Av. 0.68 Median 0.4	Annoying odor, constant pricking of mucous membranes, disturbed sleep, thirst, heavy tearing	Occupational
0.13—0.45	Burning and stinging of eyes, nose, and throat, headaches	Occupational
0.2—0.45 Av. 0.36	Irritation of eyes and upper respiratory tract, drowsiness, headaches, and menstrual irregularities	Occupational
0.13, 0.57, and 0.44	Headaches, concentration problems, dizziness, nausea, coughing, increases in recurring infections of the upper respiratory tract, and irritation of eyes, nose, and throat	Schools
~0.83	Loss of olfactory sense, increased upper respiratory disease, sub- trophic and hypertrophic alterations in nose and throat, ciliostasis of nasal mucosa, increased absorptive function of nasal mucosa	Occupational (greater than 5 years to less than 10 years)
0.9—1.6	Itching eyes, dry and sore throats, disturbed sleep, unusual thirst upon awakening in the morning	Occupational
0.9—2.7 ?	Tearing of eyes, irritation of nose and throat	Occupational
1.3—3.8	Chronic airway obstruction, respiratory tract and eye irritation, small decrease in pulmonary function during workday and work week Menstrual disorders, pregnancy complications, low birth weight of offspring	Occupational

Tolerance to formaldehyde odor may develop after several hours of exposure and repeated exposure may lead to hypersensitivity (85). In addition each effect occurs at a wide range of concentrations. As the concentration of formaldehyde increases, both the severity of reaction and the number of people responding increases.

Information regarding the effects of formaldehyde on humans comes from controlled exposure studies. Low level exposure of from 10 minutes to 5 hours under controlled conditions leads to eye, nose and throat irritation at 0.2 ppm and above (44). These effects generally disappear whenever exposure stops (5). At low concentrations the major effect of formaldehyde is irritation of the eyes and mucous membranes. Effects usually begin to appear between 0.05 and 1.5 ppm. Here non-specific complaints such as thirst, dizziness, headache, tiredness and difficult sleeping are reported.

EPA has determined irritation of eyes (burning and tearing), nose, throat and lungs occurs for the majority of people at 0.1 to 1.1 ppm (86). Eye irritation was reported at 0.05 ppm and above. Beginning at 1 ppm nose, throat and bronchial irritation occurred and it was commonly reported at 5 ppm.

Headache, nausea, coughing, chest constriction, rapid heartbeat and pressure in the head occur at 1-2 ppm (5) as well as drowsiness, vomiting and diarrhea. Concentrations greater than 5 ppm are likely to produce wheezing, coughing and chest constriction (79). At 50 ppm formaldehyde produced bronchial inflammation, pulmonary edema, pneumonia and sometimes death (85). Table 4-3 shows the concentrations of formaldehyde at which effects have occurred in controlled human and animal studies (44).

The NAS Committee on Toxicology concluded there is no population threshold for irritant effects of formaldehyde. NAS suggests most healthy adults will not experience acute toxic effects at below 0.1 ppm and something less than 20 percent will experience reactions at less than 0.25 ppm. However, individuals vary in their susceptibility. Infants, people with respiratory problems, allergic individuals and the elderly may respond at lower levels or experience more severe effects (84).

Most sensitive individuals can detect the odor of formaldehyde at approximately 0.05 ppm. A few may even experience allergic reactions at these levels. About 50% of healthy people detect formaldehyde at 0.17 ppm - a concentration which may also irritate the eyes and upper respiratory tract (81), and most people can detect 1.0 ppm (5). Thus some people may experience irritating effects at formaldehyde levels which they cannot smell.

Sensitive Individuals and Sensitization

High sensitivity to low levels of formaldehyde occurs in 10-20% of the population. Some susceptible people experience asthmatic symptoms at low concentrations (5). About 4% of the population is particularly sensitive to developing allergic dermatitis when skin is exposed (81).

In addition to its action as an irritant in both humans and animals formaldehyde also has sensitizing effects (44). Chronic exposure can heighten

Table 4-3 Lowest Effective Concentration: Controlled Studies

ppm	Length of exposure	Species	Effect
0.01	5 min	Human	Eye irritation
0.05—0.06	min	Human	Odor threshold
0.07	min	Human	Optical chronaxy threshold
0.08	1.5 months	Rabbit	Changes in evoked potential of optic nerve
0.08	min	Human	Threshold to affect the functional state of cerebral cortex
0.2	1 hr	Human	Eye, nose, and throat irritation
0.25	5 hr	Human	Dryness of nose and throat, decrease in mucous flow rate
0.31	1 hr	Guinea pig	Increased airway flow resistance, decreased compliance
0.55	10 min	Rat	Reduction in respiratory rate
0.83	3 mon	Rat	Histological and histochemical changes in cerebral amygdaloid complex
0.83	1 min	Human	Altered functional state of cerebral cortex
0.83	90 days	Rat	Peribronchial and perivascular hyperemia, lymphohistiocytic proliferation in lung, focal hyperplasia and RE system activation in cerebral cortex
0.83	10 min	Human	Irritation of upper tract and eyes, accelerated breathing, EEG changes, such as alpha rhythm enhancement, changes on automatic nervous system
0.83	10 months over 2 generations	Rat	Morphological changes in upper respiratory tract, decreased liver weight
0.83	Continuous beginning 10—15 days before mating	Rat	Increase in size and number of extramedullary hematopoietic centers, increased epithelial proliferation of common bile duct, increased abnormalities of renal epithelium
1.4	1 min	Human	Eye sensitivity to light lowered in unacclimated group
1.67	Continuous or intermittent	Guinea pig, rat	Sensitization (inhalation) leukocytosis and change in blood cholinesterase
2	6 hr/day, 5 day/week for 18 months	Rat	Epithelial hyperplasia, squamous cell metaplasia of nasal turbinates, rhinitis
3.8	90 days continuous	Rat, dog, rabbit, monkey, guinea pig	Death in 1/15 rats; some inflammation in lungs in all species
4.1	4 hr/day on days 1—19 of gestation	Rat	Increase in threshold of neuromuscular excitability, peripheral white blood cells, decreased hemoglobin, and rectal temperature in pregnant animals
4.2	1 min	Human	Unbearable without respiratory protection
5.6 and 14.3	6 hr/day/week for 24 months	Rat, mice	Carcinogenic and other pathological changes of the nose (see text)
15.5	10 hr	Mouse, rabbit, guinea pig	5/7 mice, 3/5 rabbits, 8/20 guinea pigs dead; closed eyes, slow deep respiration, convulsions
41.5	1 hr/day, 3 day/week for 35 weeks	Mouse	Upper respiratory tract inflammation, basal cell hyperplasia, epithelial stratification, bronchopneumonia
50	1 hr/day, 1 day/wk for 18 months	Hamster	Squamous cell metaplasia
482	4 hr	Rat	L. C. 50 approx. 1
0.2	Min in water	Human	Elicited allergic dermatitis response in a sensitized subject
3.7—37 g/t and 3.7 g/t as challenge solution	Intermittent patch induction	Human	Induced dermal sensitization in 4.5 to 7.8% of the healthy test subjects

(44)

sensitivity (5). Repeated exposure to liquid or vapor formaldehyde can sensitize certain individuals. When re-exposed they may have an allergic skin reaction which can include oozing and other effects, or mild or severe asthmatic reaction. Responses may increase in intensity over time. The NAS Committee on Aldehydes has indicated persons sensitized to formaldehyde and with hyperactive airways (10-12% of the U.S. population) have more severe responses (44).

Formaldehyde as a Human Carcinogen

A link between formaldehyde and cancer has not yet been definitively shown in humans. However, the Federal Panel on Formaldehyde and several Federal agencies have suggested formaldehyde be considered a human carcinogen until proven otherwise (5). This decision was based on evidence that formaldehyde causes cancer in animals and limited evidence that it causes cancer in humans. Nasal cancers in animals and limited data from human studies have led EPA to classify formaldehyde as a Group B1 probable human carcinogen.

Stewart et.al. analyzed the cause of death of over 4300 workers who were employed at 10 industrial facilities which produced or used formaldehyde between 1938 and 1965. They compared exposed workers to non-exposed workers at the same facilities and to the general population in the same counties. Results listed here are for white males who comprised 79% of the workers. Cause of death did not correlate with length or level of formaldehyde exposure when 61 causes were compared. Lung cancer incidence was higher than expected (201 cases compared to 182 expected) but did not increase with level of exposure. Excessive nasal cancers were not found nor were excesses of the same cancers found among anatomists, pathologists and embalmers - brain cancer and leukemia (87).

The above study was jointly done by NCI and the Formaldehyde Institute. When the data was reanalyzed by recalculating exposure levels, job categories and employee groupings, and controlling for certain factors, a clear increase in risk of lung cancer was found at average exposure levels greater than 0.5 ppm. In addition a suggestive (though statistically insignificant) increase in cancers of the mouth and throat was found for workers in plants which produce formaldehyde resins (88).

Acute Toxicity Studies in Animals

Formaldehyde exposure has been studied in mice, rats, guinea pigs, rabbits, dogs, cats and monkeys. Formaldehyde tends to act the same way in all mammalian species.

Exposure to formaldehyde for a few minutes to a few hours show it ranges from slightly toxic (if applied orally) to moderately toxic (if inhaled) to rats, cats and mice. Formaldehyde causes mild to moderate irritation of the skin in rabbits. Liquid formaldehyde is a severe eye irritant causing corneal injury and edema in rabbits while vapors caused tearing and discharge but no injury. Half of a group of guinea pigs became sensitized when formaldehyde was reapplied to their skin 2 weeks after an initial application (85).

Extended Studies in Animals

Animals exposed to formaldehyde for 3 to 35 weeks developed pathological tissue changes. In rats and guinea pigs repeated skin contact or inhalation led to sensitization. Exposure to high doses caused salivation, vomiting, cramps and death of test animals (44).

In one 90 day continuous inhalation study rats, guinea pigs, rabbits, monkeys,

and dogs exposed to 3 ppm all developed various degrees of lung inflammation (85). In another study groups of 25 rats were exposed continuously to formaldehyde for 45-90 days. Animals exposed for 90 days to 8.1 ppm developed respiratory and eye irritation, decreased food intake and decreased liver weight (85).

In a 13 week study by the Chemical Industry Institute of Toxicology (CIIT) 20 mice and 20 rats were exposed to 4, 12.7, 38.9 ppm of formaldehyde for 6 hours a day, 5 days a week for 13 weeks. The 4 ppm group showed no adverse effects. At 12.7 ppm decrease in weight and nasal erosion occurred in 2 rats. At 38.6 ppm damage to the nasal membranes led to termination of the experiment after 2 weeks.

Pathological Tissue Changes

Pathological tissue changes in animals caused by formaldehyde include, in addition to changes in the nasal membranes, mutations, teratogenic and carcinogenic effects. Studies of dogs and rats to determine teratogenic effects showed no gross malformations, slight increase in the percentage of stillborn puppies, slight decrease in weight gain, and some changes in lymphoid and liver tissue. Formaldehyde has led to mutations in bacteria, fungi, insects, and mouse cells (but not in whole mice). It has caused chromosome breakage and recombination in yeast, insects, some cultured cells of mammals, and in rats (44).

In another CIIT project mice exposed for 1 hour a day, 3 times a week for 35 weeks to 41 ppm of formaldehyde then exposed to 123 ppm for 29 weeks, developed non-tumorous cell proliferation. A Formaldehyde Institute study of continuous inhalation showed a similar effect in rats and monkeys (85).

Formaldehyde exposure has led to cancer in mice and rats. Early studies of the cancer inducing potential of formaldehyde in rats, mice and hamsters gave negative results but the studies were questionable because of the doses used, duration of exposure, and other factors (44).

In a study by Fischer rats were exposed to formaldehyde vapor at 0, 2, 6, 15 ppm for 6 hours a day, 5 days a week for 18 months. Thirty-six of the 120 rats developed cancer in the nasal cavities at 15 ppm. This was first study to show formaldehyde to be a potential carcinogen. CIIT reported on 1980 that nasal cancer had been found in rats exposed to 6 ppm for 24 months and mice at 15 ppm for 24 months (44).

A recent New York University study of rats supports the hypothesis that formaldehyde causes cancer in animals. Rats were exposed to 14.6 ppm of formaldehyde. After 24 months 45% developed nasal cancer, while none of the controls did. This was the same type of nasal cancer as found in the CIIT and a previous New York University study (44).

Formaldehyde may be a carcinogen when in combination with other compounds. When it was administered along with benzo(a)pyrene and diethylnitrosamine it did not increase the incidence of tumors with the former but did with the latter (44).

FORMALDEHYDE MEASUREMENT AND MONITORING

Three quantities important to measure with respect to formaldehyde are emission rate, concentration in the air and the total exposure to which an individual is exposed. Because emission rates and air concentrations change and are a function of many variables, personal monitoring at breathing level is the best method of determining total exposure. Personal monitoring however is tedious and expensive, is done occasionally in industrial situations but seldom in residential (81).

Emissions Testing

Four general types of emissions measurement procedures are utilized for products which release formaldehyde such as bonded wood products and UFFI. These are static, dynamic chamber, equilibrium chamber and distillation/extraction tests. Each is described briefly below. (See reference (83) for more details and the advantages and disadvantages of each method.)

Static tests rely on diffusion of formaldehyde gas from the sample to a medium in which it is collected. The dessicator test is commonly used and utilizes a tight glass container, a rack for samples, and a beaker or plate to hold the collection medium which is a measured quantity of distilled water (Fig. 4-2) (83). This system is maintained at 75°F for 2 to 24 hours with longer time periods used for materials with lower emission rates. The amount of formaldehyde absorbed by the water is converted to an emission rate using a calibration table.

The amount of formaldehyde emitted by a sample and absorbed by water can also be determined using analytic methods such as the chromotropic acid, purpald, acetylacetone and pararosaniline procedures.

Dynamic chamber tests pass air at a controlled rate past a sample in a chamber. Formaldehyde is carried out of the chamber with the air. This technique models air contamination in a room. Chambers may be laboratory size or large enough to hold a full size sample of a material such as particleboard.

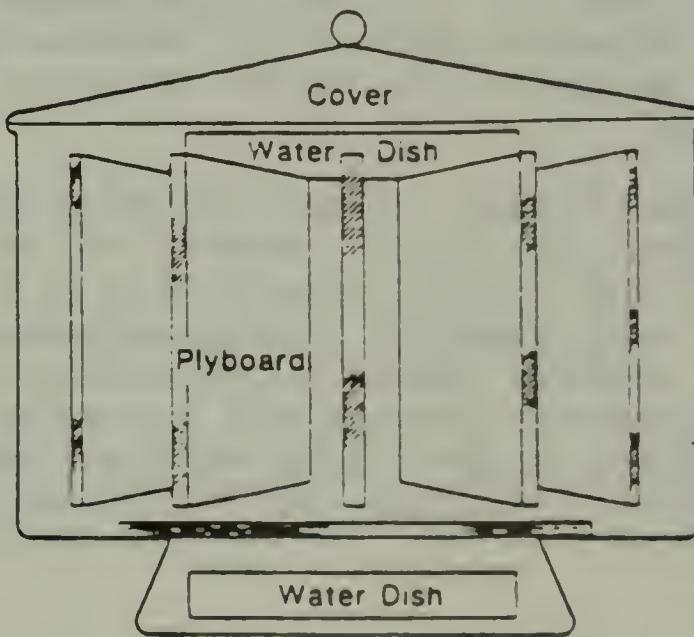


Figure 4-2.

Desiccator Test Apparatus

(83)

Equilibrium chamber tests are similar to static chamber tests. The test material is placed in a closed container without any air change. After an equilibrium is reached between the formaldehyde concentration in the material and the air, the formaldehyde content of the air is measured.

The distillation/extraction test most often used involves using a boiling solvent such as toluene to collect the formaldehyde. Samples are placed in the boiling toluene. Then the toluene with formaldehyde is bubbled through distilled water to extract the formaldehyde.

The formaldehyde surface emission monitor does not destroy the sampled matter and is portable (Fig. 4-3). It consists of a covered fine brass sieve which is placed about 2.3 cm above the test substance. A solid sorbent material is sprinkled on the sieve, and the system is sealed for 2 hours. The sorbent is then washed with distilled water from which the formaldehyde is collected. This monitor is sensitive enough to detect emissions from any substance known to affect indoor formaldehyde concentrations.

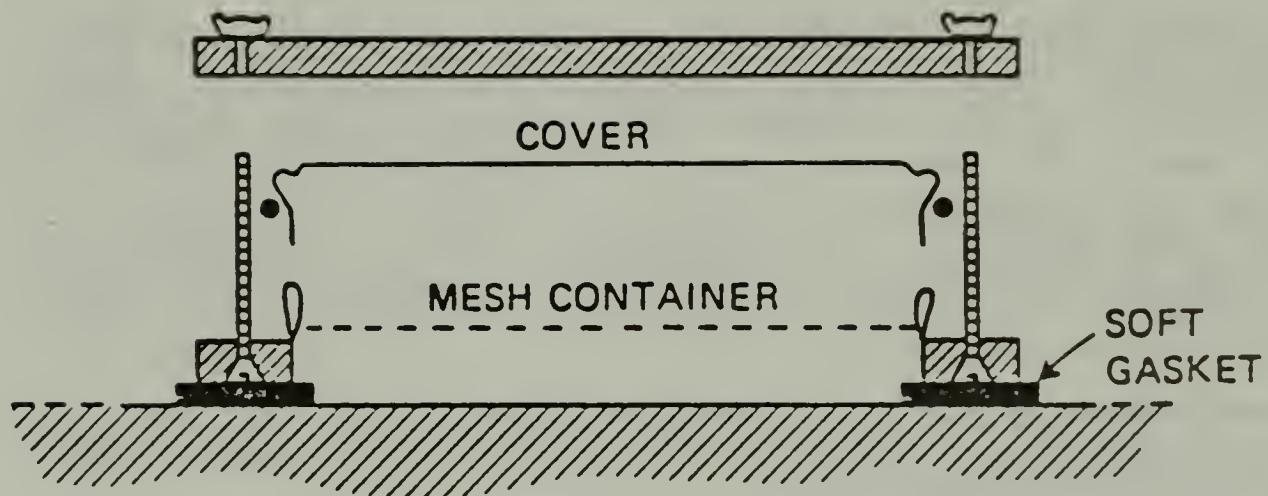


Figure 4-3

Formaldehyde Surface
Emission Monitor

Monitoring Air Concentrations and Personal Exposure

Concentrations of airborne formaldehyde can be obtained from formaldehyde surface emission monitors by determining the emission rates, the areas of various emitters, the volume of living space, and the measured air exchange rate (44).

Air concentrations are measured using the NIOSH standard method 3500 (adapted from P&CAM 125), also referred to as the "chromotropic acid" method. The working range of this method is 0.02 to 0.4 ppm for a 90 liter air sample. It is the most sensitive formaldehyde method in the NIOSH Manual of Analytical Methods, against which all other sampling methods are validated. Using this method, sampling would take 90 minutes at an air flow rate of 1 liter/minute. In addition, a passive dosimeter has been validated against the NIOSH method 3500 to +/- 25% when exposed for 168 hours (7 days), with a lower level of detection of 0.02 ppm. DPH uses the latter.

DEQE's Air Quality Surveillance Branch recommends indoor air monitoring for formaldehyde (and related aldehydes) with impinging air sampling techniques using duplicate sampling trains. It samples room air, the other functions as a reference sample (charcoal filtered air, distilled water). The air pumped through this sample train is mixed with specific chemical reagents and produces a characteristic color - change which can be measured against a standard.

The Air Quality Surveillance Branch also uses a portable toxic gas monitor wet-chemistry analyzer to perform this task. This can be set-up for indoor as well as ambient air real-time monitoring. The analytical scheme is as follows: air is sampled through a calibrated air pump built into the instrument at a rate of 0.5 liters/minute. Any aldehydes (formaldehyde) present will react with two chemical reagents, sodium sulfite and pararosaniline, to produce a colored product and measured in a photocell, yielding real-time part per billion concentrations. (Linear range of instrument is 0-250 ppb).

Information as to the presence of formaldehyde in room or ambient air can be obtained through the use of Draeger colorimetric tubes. These tubes detect pollutants by manually pumping air through the chemically reactive resin, producing a net color-change proportional to the concentration in parts per million.

Another method for sampling for aldehydes is found in the EPA Compendium of Air Toxics Methods, section TO-5. This method involves ambient air sampling using impinger samplers containing absorptive solutions, which are then extracted using solvents to isolate the formaldehyde. Formaldehyde levels are determined with high performance liquid chromatography.

Several air sampling devices are available which also may be adapted as personal sampling devices to determine total individual exposure. Most widely used is a dry sulfite device. Its sensitivity limitations require 5-7 days of exposure. A passive bubbler monitor passes formaldehyde through a permeable membrane where it is absorbed by 3-methyl-benzothiazolone hydrazone. The manufacturer indicates it can detect 0.1 ppm through a 2 hour exposure. Another which is in the development stage, contains a film of hydrobenzoic acid hydrazide which forms crystals when exposed to formaldehyde. These can be "read" visually. Pumped tube methods, which are adequately sensitive, use solid sorbents. One of these, the Tejada method, was developed by EPA and is being used in its Toxics Air Monitoring System (89).

FACTORS AFFECTING FORMALDEHYDE LEVELS

The level of formaldehyde in a room depends on the rate at which it is being emitted, and the rate at which it is being removed. Emission rate is a complex of many factors while removal is a function of ventilation or air cleaning factors. As discussed below, however, there is an interactive effect between existing formaldehyde concentration, ventilation rate and emission rate.

Formaldehyde Emission

The rate at which formaldehyde is emitted by a source depends upon the nature, age and load factor of the source, two environmental factors - temperature and humidity, and the concentration of formaldehyde already present.

Nature of Source

Materials vary in formaldehyde emission rate (5). This is due to differences in the formaldehyde compounds they contain, certain physical characteristics and their condition. Products with PF rather than UF resins emit considerably less formaldehyde. Fibrous glass insulation and ceiling tile with PF resins are unlikely to increase indoor levels by more than 0.02 ppm (83). Products which have been treated to reduce emission, e.g. by coating exposed surfaces, have lower rates (44).

Plastics which are molded, high density, extruded or non-porous release little formaldehyde (44) whereas UFFI, which is porous, emits high levels. Drapery and upholstery fabric treated with UF resins have low emission rates of only about 0.015 mg/sq m/hr which could only raise indoor levels by more than 0.01 ppm in very high loading situations.

Combustion of both petroleum and plant products releases formaldehyde. Thus formaldehyde is a product of burning fuel for cooking or heat and smoking tobacco products. For example, gas ovens and ranges can emit from less than 2 to nearly 30 mg/hr (83). In one test an oven and stove top burner emitted 25 and 15 mg/hr respectively (44). Gas space heater emissions range from less than five to over 60 mg/hr depending on burning efficiency and new kerosene space heaters up to six mg/hr. Combustion appliance data shows formaldehyde emission rate depends on whether the appliance is properly tuned and functioning. Data for sidestream cigarette smoke emissions show it ranges from 0.2 mg to nearly 1.5 mg per cigarette (83).

Age of Source

The rate of formaldehyde emission decreases as materials cure and age. Most likely half the formaldehyde in most materials is released within 2-5 years. Bonded wood products contain and emit both free formaldehyde and formaldehyde bonded into resins. Free formaldehyde is the residual formaldehyde left after the paneling or particleboard is hot pressed to cure it. Free formaldehyde can be released rapidly and most of formaldehyde initially released is of this type. Formaldehyde release is proportional to the total residual formaldehyde in a product and emission decreases exponentially with age. Generally formaldehyde release due to compounds breaking down occurs in lower amounts over a longer period. The various forms of bound formaldehyde differ in their rates of decomposition and susceptibility to reacting with water (44, 81, 83). Therefore they will differ in emission rate.

Similarly, emission rates of UFFI generally decline with time. Most UFFI installations release the greatest amount of formaldehyde in the first year (84). The Connecticut Department of Health Services surveyed formaldehyde levels in 30 UFFI homes in 1986. These were selected from 500 previously showing high levels. Ten controls without UFFI were used. Most

homes insulated with UFFI prior to 1981 had levels at or near those of the controls. A few still had levels up to 0.3 ppm. Controls averaged 0.04 ppm while UFFI homes averaged 0.08 ppm. Age of insulation inversely correlated with formaldehyde level (90). New apparel has very high emission rates - up to 0.03 mg/sq m/hr. However laundering decreases the emission rate (83).

Load Factor

Load factor is the ratio of a product's surface area to indoor air volume. The greater the surface area per unit of air volume the higher the emission rate (81). Adding new furnishings such as cabinets or furniture containing bonded wood products can increase total emissions of formaldehyde by increasing the load factor. Mobile homes often contain higher formaldehyde levels than conventional homes because more particleboard and plywood are used in their construction, i.e., they have higher load factors (5). In UFFI homes the insulation has a very high surface area and therefore high emissions.

Temperature and Humidity

An increase in temperature, humidity or both leads to an increase in formaldehyde emission. The emission rate increases exponentially as temperature rises (Fig. 4-4). This response occurs relatively rapidly. In fact emission rate has been found to track daily solar temperature variations (Fig. 4-5) (81).

As amount of humidity in the air increases, the amount of formaldehyde emitted increases accordingly. However because of the high moisture capacity of wood and the slow moisture exchange rate between air and wood, moisture equilibrium may take a long time to achieve (81). The emission response to increased humidity is thus much slower than the temperature response and is believed to be linear (83).

Formaldehyde readily combines with water to form methylene glycol and is attracted to areas where moisture is present such as air conditioning ducts, cold outside walls and areas where wood is present (wood contains water). Moisture itself enhances the release of formaldehyde from resins (81).

Concentration

The emission rate of a product is inversely related to the formaldehyde concentration in the surrounding air. Formaldehyde like any gas will move from areas of higher to lower concentration (44). Formaldehyde will be emitted into the air of a formaldehyde-free test chamber or closed room at an ever decreasing rate until equilibrium is reached. Thus, emission rate is both a component of concentration and affected by concentration.

Ventilation and Air Cleaning

Ventilation has a direct effect on both formaldehyde concentration and emission. Test chamber studies have been utilized to study the relationship

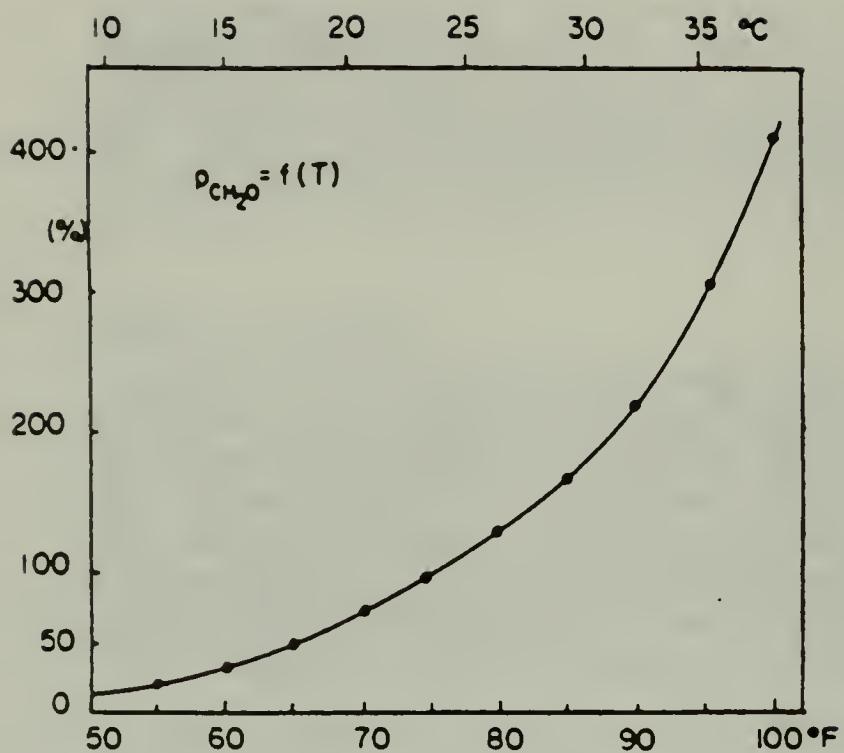


Figure 4-4. Formaldehyde Emission from Pressed Wood Products as a Function of Temperature (81)

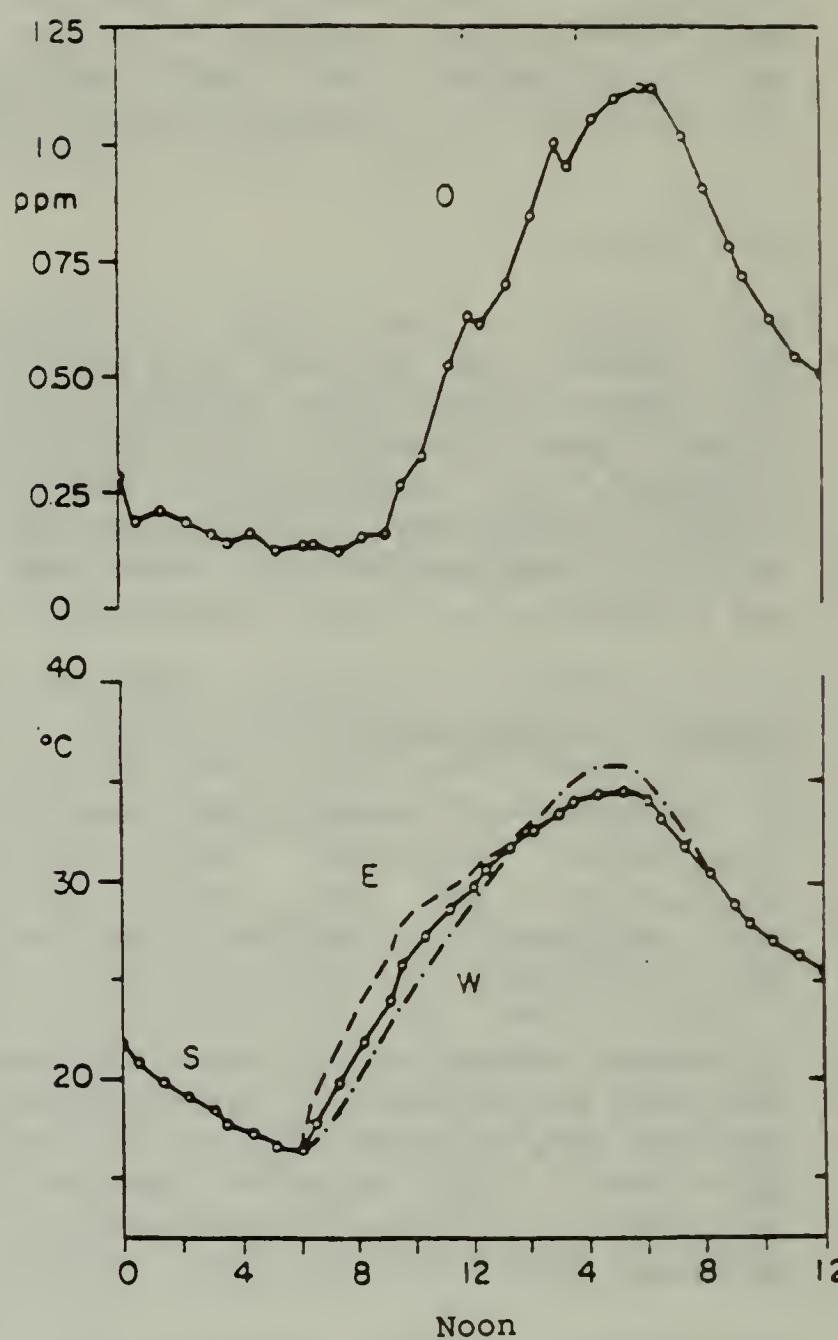


Figure 4-5.

Daily Variation of Formaldehyde Concentration and Wall Temperature in a Mobile Home. E = east wall, S = south wall, and W = west wall (81)

among these factors (44). As the number of aches increases and formaldehyde levels drop, emission rate increases (Fig. 4-6). However the relationship is non-linear (81, 83). Doubling the ventilation rate may reduce airborne formaldehyde concentration by 33-38% as opposed to the 50% increase expected. Air cleaning techniques which lower formaldehyde concentration presumably have the same effect.

Interactive Effects

An example of how formaldehyde levels are affected by emission factors and ventilation is an Oak Ridge, Tennessee study. Twenty-four hour integrated monitoring was used to assess the factors determining formaldehyde levels in 40 houses representing a diversity of age, construction type, heating and insulation type (including 11 UFFI) (44).

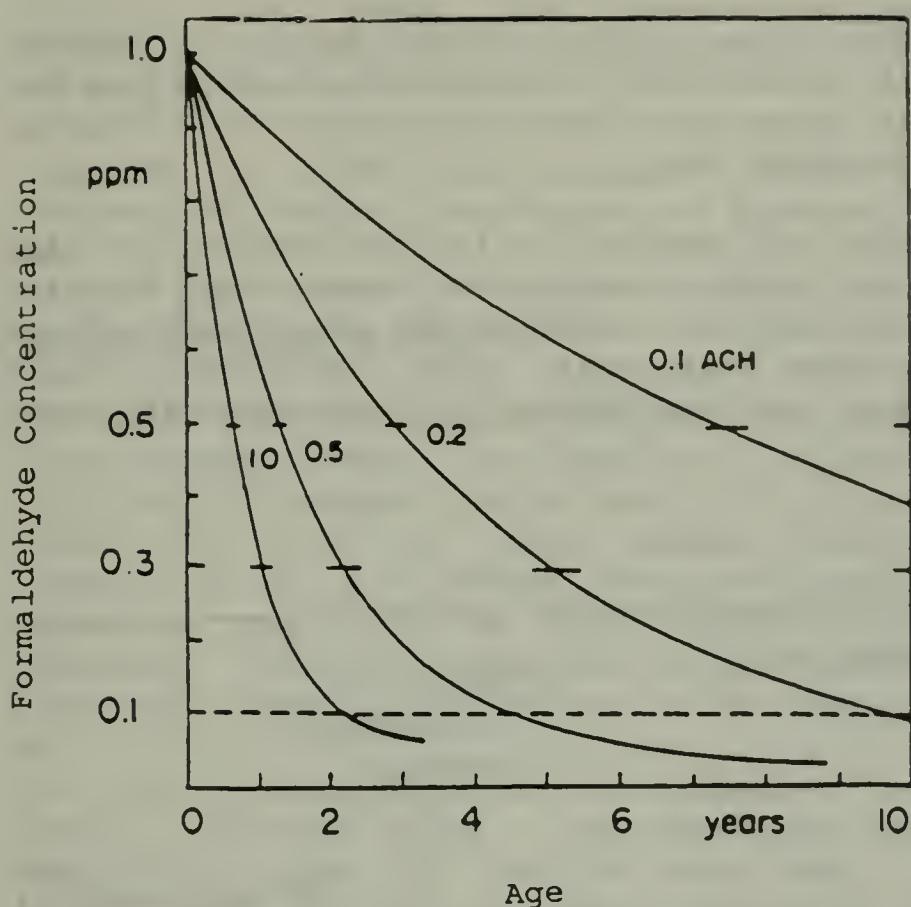


Figure 4-6.

Formaldehyde Release from Particleboard as a Function of Age and Ventilation Rate.

(81)

Indoor formaldehyde levels fluctuated with time. Daily variations occurred as a result of occupants' activities and the solar cycle. Studies of a 3 year old prefit UFFI house indicated formaldehyde levels increased in the middle of the day as the sun heated the walls. By contrast in a ten year old non-UFFI house (Fig. 4-7) maximum concentrations occurred in the early morning when the house was closed and people were asleep (44).

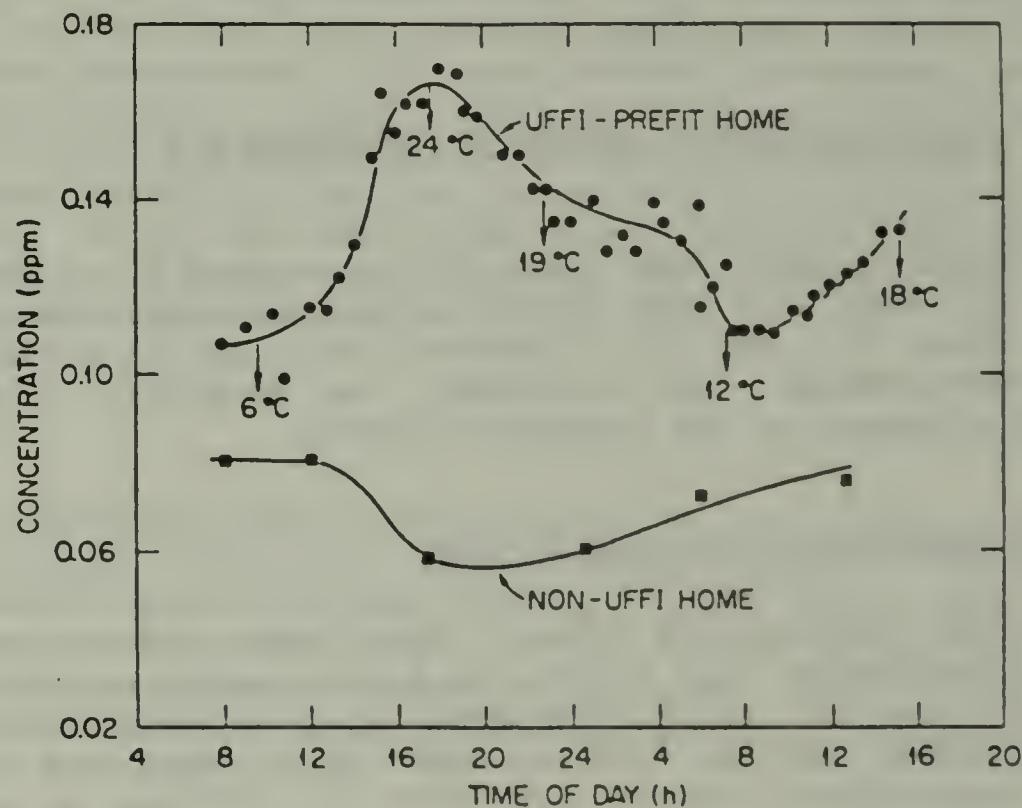


Figure 4-7

Diurnal Variation

of Formaldehyde Concentration

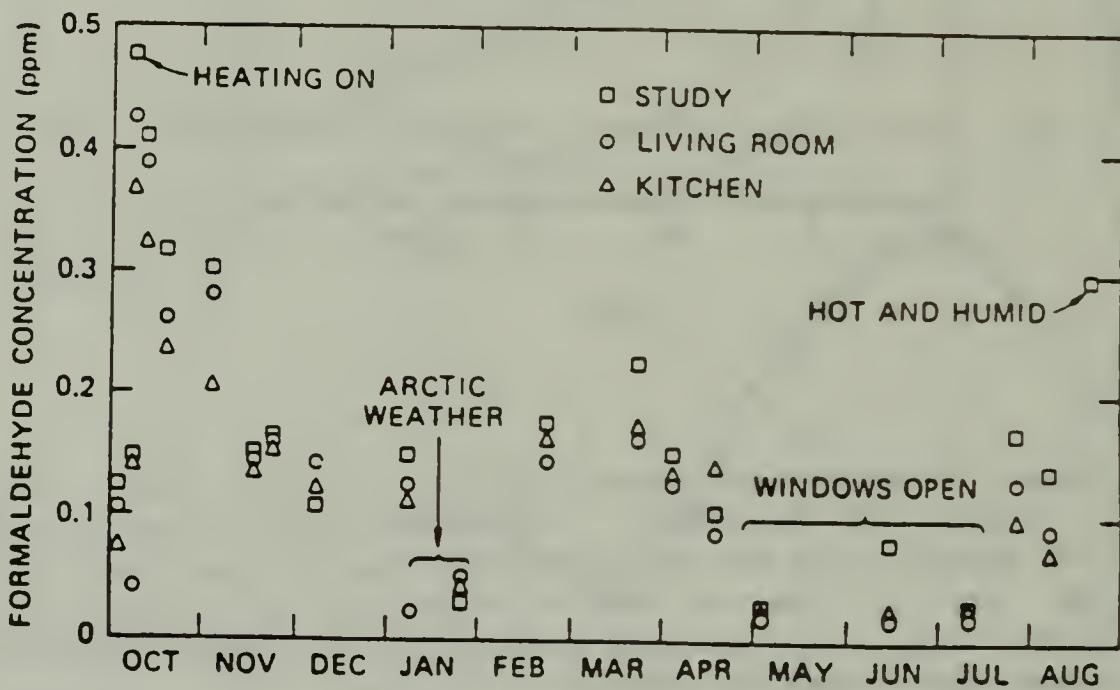
in UFFI and Non-UFFI Homes

(47)

Seasonal changes occurred in a UFFI house. In winter the drying and cooling of the UFFI (Fig. 4-8) decreased formaldehyde concentrations after they had increased when the heat was first turned on. Levels increased in the spring but opening windows then led to a decrease (44).

The interactive effects of emission and ventilation factors explain the high levels of formaldehyde found in: new tightly constructed homes (high emission rates of new materials, little ventilation); mobile homes which have a large amount of bonded wood (high emission materials, high load factor, little ventilation due to their tightness); and UFFI homes (high emission of porous material, high load factor).

Figure 4-8.
Variation of Formaldehyde
Concentration with
Time of Year



FORMALDEHYDE: SCOPE OF THE PROBLEM

Rural outdoor air generally contains 0.005-0.05 ppm of formaldehyde. Levels in urban air are almost always below 0.05 ppm and indoor levels are usually below 0.1 ppm (81). However levels may be elevated due to particular uses of formaldehyde in buildings, the materials used in or structure of the buildings, or the occupants' habits.

Laboratories and Industrial Facilities

High formaldehyde levels have been found in specialized situations such as laboratories and industrial facilities where formaldehyde is used as part of a process or as a component of a product. For example, in high school and college biology laboratories where specimens are preserved with formalin, formaldehyde levels may be 0.14 - 1.36 ppm in the parts of the lab where the students are working compared to 0.03 - 0.36 ppm in other areas of the room.

Anatomy labs where human cadavers are used range from 0.4 - 6.0 ppm. Other parts of schools and colleges generally have formaldehyde levels of less than 0.05 ppm (44).

Surveys by NIOSH over the past 10 years indicate formaldehyde levels to which workers in various industries are exposed (Table 4-4) (44).

Building Contents or Structure

By 1983 3 billion square feet of particleboard was being produced each year in the United States, as well as 2 billion square feet of hardwood plywood and 600 million square feet of MDF (83). Around 300,000 metric tons of UF resin a year are used for these pressed wood products. Most buildings now contain from 0.2 to 1.0 square meter of product per cubic meter of air. UF bonded products are now the most abundant formaldehyde emitting products in North America. Melamine formaldehyde bonded plywood is not used much and PF bonded exterior grade plywood emits little formaldehyde (81).

In the Tennessee study formaldehyde was measured every other week in three rooms of each house. In spring and summer 22 of the 40 homes had readings above 0.1 ppm, at one or more times. Those were mainly the UFFI homes and homes aged 5 years or less. Thus many residences may at least at times have levels greater than the 0.1 ppm ASHRAE ceiling (44).

Because the bulk of formaldehyde is emitted in the first year or years, and because conventional homes are not tightly constructed, older conventional homes seldom have formaldehyde problems. However tighter, newer buildings resulting from the energy crisis of the 1970's have lower exchange rates between indoor and outdoor air, and generally higher indoor formaldehyde concentration. Formaldehyde levels of over 0.1 ppm may occur due to low infiltration rates (44).

The best data for exposures in conventional homes is obtained by random sampling of those built after 1982 when lower emission resins came into use. Studies by Lawrence Berkeley Laboratories, CPSC, the Canadian government, and state governments and universities in Iowa, Indiana, Texas and California suggest the average level in conventional homes is currently around 0.05 ppm. Means of the studies range from 0.03-0.09 ppm. Newer and energy efficient homes have levels from 0.1 to 0.2 ppm while homes over 5 years old average 0.005 to 0.08 ppm. Comparison of these results with data from studies prior to 1980 indicate little change in conventional home levels (83).

Mobile Homes

Problems with elevated formaldehyde levels have occurred in office trailers, temporary or portable school buildings and mobile homes. Common characteristics of these structures are surface loading factors over 1 square meter of UF bonded products per cubic meter of air, poor insulation and poor ventilation. Such buildings are often located in open areas where sun and wind can cause the buildings to reach humidity and temperature levels which compound formaldehyde release problems (81).

Mobile homes present the highest exposure to formaldehyde of all types of dwellings regardless of age (44). Formaldehyde concentrations were often found to exceed 0.1 ppm in Denmark, the Netherlands, and the Federated Republic of Germany (10). Over 200 mobile homes assessed in Washington state after occupants complained had 0.03 - 2.4 ppm, with similar findings in Wisconsin (7). Median concentrations of 0.1 ppm were found in 100 mobile homes in Texas and 0.58 ppm in Minnesota units where residents complained (81).

Mobile homes built after 1982 were also assessed in the studies by Lawrence Berkeley Laboratories and others discussed above. Mobile home levels have decreased since lower emission products have been in use. If complaint homes are included levels in individual homes range from less than 0.1 to 1.0 ppm. Average levels are now 0.2 - 0.5 ppm (83).

UFFI Homes

After mobile homes the highest formaldehyde concentrations are found in homes with UFFI, again regardless of age. UFFI homes have been found to contain levels four times as high as their conventional counterparts, which seldom have high levels. Use of UFFI increased throughout the 1970's. UFFI was installed in 435,000 residences between 1975 and 1981 and was estimated to be in 500,000 American homes by 1983 (44,84).

Data collected by Cohn et.al. from many studies of UFFI homes show that formaldehyde levels decrease as the UFFI ages. However even after 9 years the average reading is above 0.1 ppm (Table 4-4). In this study each of the 1164 points represents the average reading in a UFFI home - mostly measured by the chromotropoic acid method. The most rapid decline was in the first 40 weeks. Other studies have indicated the range of concentrations of formaldehyde in UFFI homes to be from 0.01 to 4.1 ppm with an average of 0.14 ppm (83).

Occupants' Living Habits

Smokers expose themselves and others to formaldehyde produced by combustion. A smoker of a pack of cigarettes a day is exposed to 0.38 mg of formaldehyde. Non-smokers are also exposed to this formaldehyde (44).

Whether cigarettes affect indoor air levels significantly depends on the occupants' living patterns and ventilation. In studies where 10 cigarettes were smoked in a home each day formaldehyde levels did not increase significantly over controls. Studies where a number of people chain smoked in a poorly ventilated room showed quick increases in formaldehyde levels (83). For example, in a study where 5 people smoked a total of 20 cigarettes within 30 minutes in an average size room the formaldehyde level rose from 0.01 to 0.27 ppm. Undiluted cigarette smoke contains up to 40 ppm (by volume) of formaldehyde (44).

Stoves and heaters are also significant emitters as discussed in the previous section. Clearly formaldehyde levels above 0.1 are occurring today not only in mobile homes and UFFI homes but also new conventional and energy efficient homes, and may occur in any home if heavy smoking is occurring.

Table 4-4

Formaldehyde Concentration in UFFI Homes

Days	Number of data points	Avg ppm
0-71	63	0.210
141-210	76	0.240
211-280	51	0.240
281-350	58	0.058
351-420	72	0.068
421-490	55	0.084
491-560	68	0.100
561-630	45	0.076
631-700	49	0.078
701-770	70	0.080
771-840	37	0.081
841-910	54	0.079
911-980	45	0.058
981-1050	44	0.082
1051-1120	66	0.072
1121-1190	30	0.050
1191-1260	46	0.040
1261-1330	29	0.072
1331-1400	22	0.054
1401-1470	22	0.074
1471-1540	8	0.063
1541-1610	6	0.047
1611-1680	15	0.032
1681-1750	9	0.021
1751-1820	14	0.067
1821-1890	4	0.102
1891-1960	5	0.039
1961-2030	4	0.080
2031-2100	5	0.054
2101-2170	4	0.050
2171-2240	4	0.078
2241-2310	1	0.040
2521-2590	1	0.117
3011-3080	2	0.030

(83)

RISK ASSESSMENT FOR FORMALDEHYDE EXPOSURE

Hazard Identification

The irritant properties of formaldehyde have been substantiated through animal studies and both occupational and controlled studies in humans. EPA's risk assessment concludes that human exposure to formaldehyde can lead to acute effects at 0.1 to 1.1 ppm. These effects include irritation of the eyes, nose, throat and lungs. Changes in the cell structure of the nose may occur within the same range. Formaldehyde is mainly deposited in the upper respiratory tract including the nasopharynx (89).

Mutagenic, teratogenic and carcinogenic effects have been produced in animals exposed to formaldehyde. Cancers of the nasopharynx, in particular, can be due to formaldehyde. Exposed rats have developed nasal cancer, which is very rare in unexposed individuals (89). Elevated incidence of nasal cancer has been found in rats exposed to high concentrations of formaldehyde vapors in the 1980 CIIT study, and reinforced in studies by New York University and by Albert and Tode in Japan (33).

Limited human data also suggests that formaldehyde exposure might lead to cancer. Retrospective cohort studies have not shown excess nasal cancer (89) but some have shown significant increases in other cancers (87).

Dose Response

The type of acute health effect produced by exposure to formaldehyde varies with concentration. A given effect appears within a range of concentrations, and within the range of response the fraction of the population experiencing the response increases.

In the CIIT study a 2-3 fold increase in formaldehyde exposure led to a 50% increase in tumors (33).

A model which is often used assumes that cumulative exposure rather than dose alone determines risk, that formaldehyde affects the cells it initially contacts, and that formaldehyde identified at specific sites in the body is directly proportional to the amount inhaled. There is some evidence that the linear model may be inappropriate (33).

Exposure Assessment

It is difficult to determine how many people are exposed to formaldehyde and what proportion of these have adverse health effects. There is evidence that a substantial fraction of exposed people do have effects (85). The fact that irritant effects occur indoors makes it clear that exposures which create acute effects are present.

People may be exposed to formaldehyde in both indoor and outdoor air from

occupational sources, building materials and consumer products. An estimated 1.4 million people are exposed in occupational settings. The exact exposures in each industry are not known but are probably less than 3 ppm and more likely 1 ppm (85).

Eleven million people live in homes with either UFFI or particleboard made with UF resins. Concentrations from 0.01 to 10.6 ppm occur, though most are less than 0.05. Since most people spend up to 70% of their time indoors total exposure could be high. Outdoor concentrations seldom exceed 0.1 and are usually less than 0.05. Exceptions occur where there is unusual fuel use or traffic density (85).

Asthmatics may be more susceptible than others and have acute reactions even at low concentrations. An estimated 10-20% of the 220 million people in the United States may be hypersensitive to low concentrations (above 1.5 ppm) (93). A number of acute symptoms are associated with formaldehyde exposure (89).

Exposed populations in both occupational and residential settings have been reported to have asthma and diminished lung function - both acute and chronic. For example, in a comparative study of people living in UFFI and non-UFFI homes, those in UFFI homes had significantly more wheezing. In addition a fraction of the population has severe allergic response to elevated indoor formaldehyde levels and formaldehyde has been associated with occupational asthma (89).

Some occupational studies show elevated levels of various forms of cancer. However, the actual levels to which workers were exposed is often unknown and therefore, it is not clear whether the concentrations which produced nasal cancer in rats occur in the workplace. In addition it is still not clear whether total dose or exposure is the determining factor in cancer risk.

EPA's priority review was triggered by the exposures found in new conventional and manufactured homes containing construction materials made with UF resins and by exposures associated with treated fabrics.

Risk Characterization

The risk of adverse health effects is related to the nature of the product present, its formaldehyde release characteristics and environmental factors (81). Assuming that animal data can be extrapolated to humans the risk of cancer for a given individual is small. For example, the additional risk after 9 years in a UFFI home is estimated at about 1 in 20,000. Individual risk will vary with actual formaldehyde levels and the length of time a person has been living in a home since as foam ages formaldehyde emissions decrease.

EPA's most recent assessment of formaldehyde's health effects classifies it as a Group B1 probable human carcinogen - based on "sufficient" evidence from animal studies and "limited" evidence from human studies (86).

FORMALDEHYDE MITIGATION

Mitigation of high indoor airborne formaldehyde concentrations is approached through elimination of the emission source, alterations or design improvements to products which reduce emissions, or through removal of formaldehyde from the air. While most mitigating techniques decrease formaldehyde levels, their success varies. Their long term effectiveness and effect on adverse health effects are unknown. Approaches vary in cost, effectiveness and ease of implementation. They may be used alone or in combination (44).

Source Control

Source elimination is theoretically an effective means of mitigation but may be costly, difficult to achieve, or lack feasibility. UFFI removal involves tearing down interior or exterior walls. Replacing bonded wood products in floors, walls, built-in cabinets requires major construction work. Replacing furniture can be a major expense. In many cases substitute products are not available or practical.

Techniques which have been used to reduce formaldehyde levels through emission control include emission barriers, chemical treatment, change in product composition, and treatment at the factory. Sealing interior wall entry points has been used in UFFI homes. Ammonia has been sprayed inside of both UFFI and non-UFFI houses to lower formaldehyde concentration. Painting or otherwise coating an object can retard emission. Some vapor barrier paints are manufactured (44).

Emissions from biological specimens can be reduced by rinsing followed by immersion in a low or no formaldehyde medium. This treatment has reduced formaldehyde levels in laboratories several fold. Time may also be regarded as a mitigant since the most reactive components of a product which generates and emits formaldehyde are usually "exhausted" first. Decreased levels over time have been found in both UFFI and non-UFFI homes (44).

Elevated indoor formaldehyde levels may be due to defective or improperly installed products, e.g., a single panel of raw bonded wood or a poorly vented stove. Mitigation is straightforward in these cases.

Compressed Wood Products

The best method of emission control is control at the site of manufacture of bonded wood products. Emissions from have been reduced by changing resin composition, adding scavengers to the wood before pressing, adding urea or wax solutions to the wood finish, fumigating with ammonia while the wood is still hot, and other methods (81).

Since there are 2 types of emissions - long term hydrolysis and decomposition and short term release of free formaldehyde - controls may reduce one or both types and sometimes decrease one while increasing the other.

Low formaldehyde resins have now decreased the residual formaldehyde content of products by a factor of 10 (81). During the 1970's resin was often produced using a 2:1 ratio of formaldehyde to urea. This has now been reduced to around 1.2 to 1.5:1 through use of different resins and has led to a decline in emissions because less excess formaldehyde is present. In the early 1960's when the first conferences on formaldehyde reduction occurred in Germany, some resins contained 1-6% free formaldehyde by weight. This percentage has been reduced by a factor of as much as a million (81,83).

PF emits relatively small amounts of formaldehyde and isocyanate resins contain none (83). These resins are of minor concern.

Emission barriers both reduce emission rate from the pressed wood and reduce the amount of water the product uptakes. Scavengers which combine with and bind the free formaldehyde are being added to UF resin-wood systems. These decrease short term emissions but their effect on long term emission is uncertain. Some coatings have scavengers which react with formaldehyde. Spraying the surface with a scavenger or exposing it to reactive gaseous ammonia has been reported to reduce early emissions by up to a factor of 10. Painting wood surfaces, adding other coatings or vinyl veneers and decorative overlays inhibit emissions to varying degrees. Some paints, for example, prevent 98% of emissions and some wallpapers prevent around 30% (83). Gypsum board and carpets also act as barriers (81) although gypsum may lead to other indoor air problems.

Before low emission products were in common use chemical remedial methods such as ammonia fumigation were used. Ammonia reaches most of the emitters except combustion sources (91) but results are often unreliable (81). By mid-1984 50% of the UF bonded wood products made in the United States met HUD requirements for mobile homes i.e. less than 0.2 ppm for plywood and 0.3 ppm for particleboard when tested in large air chambers at 25 C, 50% relative humidity, load factor of 0.94 sq m/cu m of air for plywood and 0.42 for particleboard, and 0.5 ach (81).

UFFI

The National Research Council of Canada has recommended a technique to lower formaldehyde levels in UFFI homes. Walls should be sealed through: repairing all holes, cracks or gaps with caulking or spackling compounds; applying two coats of vapor-barrier paints or mylar, vinyl or good grade canvas backed wallpaper (84). However, painting and papering may not be enough to keep formaldehyde from entering living areas. Junctions of walls and floors should be caulked with butyl or acrylic latex sealants or sealed with weather stripping or foam-backed tape. Applying a sealant such as varnish to joints and surfaces may help (84).

High concentrations of ammonia may be pumped into the house. This can be hazardous and should only be done by trained personnel. It is unclear whether low ammonia concentrations are effective. Ammonia also corrodes brass electrical and gas fittings and connectors (84).

Canada considers removal a costly method of last resort. There have been some

cases where UFFI was removed but health effects have persisted, possibly due to improper removal (84). The removal procedure calls for removing interior or exterior wall panels or siding, removal of the UFFI, and addition of new insulation and siding. Any wood surfaces which were in contact with the UFFI are treated with a stabilizing agent such as sodium bisulfite. In 1983 costs of removal were running from under \$5000 to \$20,000 with an average of around \$6000 (84). Several patents concerning UFFI claim additives to reduce formaldehyde emissions to very low amounts or zero. However tests to support this seldom take sufficient account of the effects of suppressed emission (44).

When 15 Wisconsin homes variously treated by mechanisms such as sealing or removing particleboard, and venting or removing UFFI formaldehyde levels decreased significantly compared to the levels in 15 untreated houses (44).

Removal

Increased ventilation is the only mitigation technique which is not an emission control (83). As a result of the complex interaction between formaldehyde emission rates and airborne formaldehyde concentration ventilation is not as effective in reducing concentration on a short term basis as it is with other pollutants.

Formaldehyde emission rate is determined by the difference in pressure or concentration between the emitting product and the indoor air. Ventilation lowers the airborne formaldehyde concentration. This increases the pressure difference leading to greater formaldehyde emission.

As a result of the increased emission or "outgassing", the dilution curve for formaldehyde indicates a reduction in airborne concentration of 33-38% rather than 50% for every doubling of ventilation rate (ach). It should be noted that since a product contains a finite amount of formaldehyde, increasing the ventilation rate accelerates the aging process.

Levels of formaldehyde have been reduced from 0.5 to 0.1 ppm by filtering air of mobile homes through a bed of alumina pellets impregnated with permanganate. A heat pump in its cooling mode may be effective since moisture condensing onto and water drying from the cooling coils may remove formaldehyde vapor. Forced air ventilation or pulling air through an air cleaner can be used. The effectiveness of portable air cleaners and the lifetimes of their 'sorbent' cartridges has not been tested (44).

In moderate climates where heating and cooling of homes are intermittent there may be incomplete air mixing leading to pockets of high formaldehyde concentration. Examples are paneled rooms, display shelves and areas with cabinets such as kitchens recently remodeled. In residences or commercial buildings with forced air heating or air conditioning especially in severe climates where these systems are in use much of the time air mixes and pockets of high formaldehyde are less likely to occur especially in severe climates (81).

NASA has ongoing research regarding removal of pollutants from indoor air.

Chamber studies were conducted with more than a dozen common houseplants. When high levels of formaldehyde were introduced, after 24 hours all plants reduced formaldehyde levels. The elephant ear philodendron, golden pothos and spider plant each removed at least 80% of the formaldehyde present and the aloe vera 90%. Plants may be able to metabolize, i.e., breakdown and utilize, formaldehyde (92).

FORMALDEHYDE: FEDERAL INITIATIVES

Federal action to reduce indoor formaldehyde levels has taken the form of air quality and product emission standards and, in the case of UFFI, a ban on sales.

Air Quality Standards

OSHA considers formaldehyde a potential occupational carcinogen. Over 200,000 workers are estimated to be exposed in laboratories, the funeral service and apparel industries (93).

OSHA in 1985 announced its intent to lower its 3 ppm workplace standard for airborne formaldehyde to 1.0 or 1.5 ppm. Because of this EPA's regulatory investigation for workplace exposures was delegated to OSHA (86). NIOSH recommended that OSHA's 3 ppm 8 hour time weighted average be reduced to 1 ppm (82). In late 1987 OSHA reduced the 3 ppm standard to 1 ppm (93).

A number of standards have been developed based on formaldehyde's irritant nature. Carcinogenic effects of formaldehyde exposure in animals has not been a major consideration. ASHRAE has adopted a guideline of 0.1 ppm for indoor air which it suggests provides a comfortable environment for most exposed people but this is not a guarantee of health protection especially for sensitive individuals (44). The American Industrial Hygiene Association (AIHA) also recommends a 0.1 ppm guideline. In 1978 the Netherlands established 0.1 ppm as an indoor standard for maximum permissible concentration. West Germany, Sweden, and Denmark are considering similar standards (82). However, NAS has reached the conclusion that the vast majority of healthy adults would not suffer symptoms of irritation at 0.25 ppm (5).

Product Standards

In May 1984 EPA initiated a priority regulatory investigation of formaldehyde under TSCA secs. 4f & 6 focusing on resins used in pressed wood construction materials and formaldehyde in permanent press clothing (86).

HUD has no emission standard for formaldehyde products used in houses in general (5). However, in August 1984 HUD changed its Manufactured Home

Construction and Safety Standards to limit formaldehyde emissions in mobile homes. This is a product standard which limits the level of formaldehyde from interior plywood, particleboard, floor decking and cabinets. Emissions must not exceed 0.2 ppm from plywood or 0.3 ppm from particleboard in a chamber test. Products must be certified as meeting the standard. Ambient levels in manufactured homes built with such materials are not expected to exceed 0.4 ppm at 77 F and 50% relative humidity and 0.5 ach of outdoor ventilation. This is roughly twice the actual average measured rate for new mobile homes (86).

UFFI Ban

CPSC began an investigation of formaldehyde use in consumer products in 1978. Because formaldehyde emissions were considered "too uncontrollable" to design a product standard and due to its potential risk as a carcinogen. CPSC banned UFFI in schools and residences in 1982. Canada had banned UFFI sales late in 1980 (44,86).

After the UFFI ban was issued a number of lawsuits were filed asking that it be modified or set aside. In April 1983 an opinion was issued by the U.S. Fifth Circuit Court of Appeals to set aside the ban and therefore allow sales of UFFI. Its reasoning was that the CIIT study and the home measurement and laboratory test data used to estimate formaldehyde levels in UFFI homes were not an adequate basis for estimating cancer risk. CPSC was also asked to quantify the severity of acute symptoms of UFFI exposure and the number of persons affected. CPSC countered with a petition for a rehearing indicating that the New York University study confirmed the CIIT results, its lab tests were valid and its measurements of levels in homes were from what was, "for all intents and purposes", a random sample. A second opinion in June 1983 corrected some factual errors but denied the petition (84).

UFFI has virtually disappeared from the commercial market in the U.S. and Canada (81). CPSC is investigating formaldehyde emitting products and pursuing national consensus standards reducing emissions in products made from pressed wood (86).

FORMALDEHYDE: MASSACHUSETTS INITIATIVES

Between 1970 and 1981 UFFI was installed in about 7000 homes in Massachusetts. DPH banned UFFI in November 1979 because of many documented acute health problems. At the time of the ban DPH developed a repurchasing program with regulations requiring UFFI removal at industry expense. The formaldehyde industry challenged the regulations in court which were ultimately declared invalid by the Supreme Judicial Court.

In 1985, with the cooperation of DPH and industry UFFI legislation was passed. Chapter 728 of the Acts of 1985 (effective July 1986):

- o Established an air testing program for any homeowner with UFFI installed prior to December 31, 1980.

- o Established a removal program for homes where levels of formaldehyde are greater than 0.1 ppm or an occupant has experienced adverse health effects and meets certain documentation criteria.
- o Established a trust fund supported by contributions from industry members to pay for air testing and removal. Those who receive payment for UFFI removal cannot take legal action against any industry member who has contributed to the fund. Industry members who make a "significant" contribution to the fund are exempt from the repurchase regulations.
- o Directed DPH to promulgate regulations regarding approval of testing labs, removal methods, approval of removal contractors. These regulations (105 CMR 651.000) became effective in December 1986. The DPH removal program includes a detailed manual describing the problem and corrective measures designed for use by contractors and by the DPH as material for its course for both contractors and do it yourself homeowners who wish to qualify to carry out the measures under the UFFI program.
- o Established a UFFI hotline (tollfree 1-800-222-UFFI).

- o Determined that homeowners, landlords, bankers and realtors would not be liable for UFFI-related health problems if disclosure is made by seller to buyer or landlord to tenant. Sellers and landlords must determine whether the house has UFFI. Banks and realtors cannot discriminate against homes with 0.1 ppm of formaldehyde or less.

Massachusetts has established a 0.1 ppm action level, though it recognizes homes with concentrations well below 0.1 may adversely affect occupants. DPH and DEQE believe this level should be lower.

The air testing protocol involves hanging vials in two rooms of the house, other than the kitchen or bathroom where levels would be predictably higher. They remain in place for 7 days then are returned to DPH for analysis. If the reading is over 0.75 ppm further testing is done. The initial test costs \$13 for the kit and \$22 for analysis. The second test takes 90 minutes and costs \$150-\$240 depending upon the location of the home. Currently analysis is done out of state since laboratories are selected based on competitive bidding. About 2.5% of the homes test above 0.1 ppm. The average of UFFI houses is about 0.05 ppm. 2000 homes have been tested. DPH has thus far authorized removal from 150 homes at an average cost of \$23,500.

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